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PSYCHOSOMATIC SURGERY*

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"THE OPERATION" is and will no doubt continue to be the dominant feature of surgery. In the anticipation of an operation a patient presumably has fear and dread. An operation often produces such an impression on a patient's memory that he may for the remainder of his life refer to past events as happening before or after "my operation."

The surgeon of today, as of yesterday, must of necessity be a man endowed with the sort of mind suitable for making quick decisions and proceeding with a minimum amount of delay in bringing to a conclusion a task with which he is often unexpectedly confronted. Many of his problems are of a nature which does not permit leisurely reflection. If a patient is admitted to the hospital with an acute attack of intra-abdominal disease, a decision must be reached quickly as to whether the patient is to be submitted to operation or not, and often in deciding to delay operation, the surgeon must assume an even greater personal responsibility for the preservation of the life of his patient. In the operating room, whether it be the head, chest or abdomen which is open, the surgeon is confronted with the necessity of bringing the operation to a successful or unsuccessful conclusion in a limited space of time; but no matter how objective the mind of a surgeon, I imagine

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there is none who has become so calloused that his heart does not beat faster before beginning a formidable surgical operation, or during the course of an operation when it is noted that the patient's breathing becomes shallow, the arteries spurt with less vigor and the veins become distended with dark blood.

The well known scene in the operating room has not greatly changed since the last generation. There is much the same operating team, gowned and masked and gloved. There have been no significant changes in the tools which the surgeon employs. There is the same prostrate patient, of whom Doctor Halsted spoke as an individual left with but a single weapon, his spurting artery, to resist the continued application of the therapeutic procedure. I am sure that no surgeon of today privileged to witness the manual dexterity in anatomical dissection of the masters of surgery of past generations would for a moment entertain the idea that this feature of "the operation" has improved.

Although the scene in the operating room remains much the same today as it was 25 years ago, new methods to be employed before and after the operation have made it possible not only to perform the surgical operation with far less risk, but have extended the usefulness of operative surgery for the relief of conditions previously unknown or believed to be irremediable. It is the purpose of this paper to inquire into the effect of this advance in knowledge in surgery in the realization of the fundamental purposes of surgery; that is, the prolongation of life, the removal of disabilities and the relief from an unfavorable balance in the pain-pleasure principle in those individuals receiving surgical treatment.

In the beginning surgery was objective, anatomical and for the most part destructive. Most of the operations were amputations, ligation of arteries or excision of tumors. Surgeons received their preliminary training in anatomical laboratories. The immediate result of the discovery of methods of anesthesia and asepsis was only to increase the number and scope of operations based on factors almost purely anatomical. I remember less than 25 years ago, after I had completed a gastrojejunostomy, the referring physician remarked that it was indeed a strange sort of an operation because nothing was removed. I can recall, also, when I was given the responsibility of establishing a surgical pathology laboratory for the Washington University Medical School in 1912, the very large number of normal uteri, small cystic ovaries, acutely inflamed fallopian tubes, kinked appendices and tuberculous lymph nodes brought to me for pathologic examination.

The introduction of the use of the roentgen ray served at first almost entirely to enlarge the opportunities for the determination of anatomic abnormalities. The preponderance of a materialistic philosophy in surgery is illustrated by the frequency with which surgical operations were performed for the replacement of such structures as the stomach and large bowel, which from roentgenologic examination had been presumed to be dislocated. Also, the introduction of the use of the roentgen ray in the treatment of fractures not infrequently led to a state of mind which is well illustrated by a personal

experience. An orthopedic surgeon asked me if I would come to a Roentgenologic Department so that I might see the good results he had obtained from a series of fractures of the leg. I am quite sure that he did not understand me when I told him that I would much rather see his patients walk, and it has often occurred to me since to wonder if the treatment of fractures of the leg were not more successful when it was the purpose of surgeons to make the external appearance of the fractured leg correspond as nearly as possible to that of the unfractured leg, than at the present time when surgeons attempt to secure perfect anatomic restoration of the bone fragments by the use of one or more of the several mechanical appliances which sometimes remind me of an exhibit which I once saw of the instruments used for torture at the time of the Spanish Inquisition.

During the past 25 years, and particularly during the past ten years, there has been a tremendous advancement in knowledge applicable to surgery which can perhaps best be said to belong to physiology. It is much to the credit of surgery that a great deal of this valuable knowledge has been the result of the laboratory experiments of a group of surgeons now arrived at their maturity, and who, I am glad to say, have been more than welcomely received by the older group of surgeons who did not have the good fortune of the opportunity and the stimulus for laboratory investigation.

Following the development of knowledge of the physics and the chemistry of respiration, there was almost immediately a tremendous advance in surgery of the chest. I believe I am not making any overstatement when I say that the low operative mortality and the smooth postoperative course of a patient after a pneumonectomy still seems almost miraculous to those of us who began the practice of surgery as long as 25 years ago. Recent developments in the knowledge of the physics and chemistry of the body fluids not only have reduced greatly the risk of old operations, but have made it possible to undertake with safety operations previously considered to be associated with a prohibitive operative risk. The beneficial effects of surgery have thus been extended to a large group of people previously considered inoperable. The significance of this achievement becomes greater when it is realized that our population is growing older and that individuals are not so frequently "too old" to be operated upon.

It is worth while calling attention to the fact that although surgeons have become physiologists as well as anatomists, the essential philosophy of the surgeon is still for the most part materialistic. Water, sugar, sodium chloride, proteins, carbon dioxide and even hydrogen ion concentration are just as real as muscle or tendon or nerve. There is, however, a fundamental difference between the old anatomical surgeon and the modern physiological surgeon, in that the latter has opportunities for being what, for lack of a better term, we might call constructive. An appendix can only be removed. Sodium chloride may be either added or subtracted. I believe that there can exist at the present time no reasonable doubt that the accomplishments of the surgeon have been vastly increased because of this knowledge of conditions in the human body not immediately discernible to any of the special senses.

It remains, however, to call attention to the fact that a purely materialistic philosophy in surgery carries with it the necessity of a fundamental conception which I believe is apparent in nearly all of the innovations in surgery, as well as in the evaluation of the results of the numerous well established operative procedures. The surgeon with only a materialistic philosophy must conceive of each human being as compared to a standard human being, a sort of John Doe. During the early anatomical phase of surgery, Mr. X, who differed from John Doe, had only the possibility of surgery making him into a conventional part of John Doe. The addition of the physiologic concept into surgery brought with it the idea that certain departures of a Mr. X from John Doe might be restored or compensated for, in order to improve Mr. X's well being, or, more frequently, to improve Mr. X's chances of survival of some major anatomic alteration.

I have had the good fortune to observe a considerable succession of men through periods of five years of postgraduate training in surgery, and I am quite sure that I am correct in stating that almost all the effort of the Resident Surgeon today is directed toward the accumulation of facts concerning abnormalities of anatomy and physiology of the patients for whom he is trusted to assume great responsibility. No other than the most arrogant and selfish Chief Surgeon would fail to recognize the enormous amount of comfort he derives from the knowledge that an efficient Resident Surgeon with only a materialistic philosophy is in constant attendance on the surgical wards, not to mention the number of times success has been obtained or even a catastrophe has been averted in the Chief's own cases by prompt action or timely suggestion of the realistic resident.

It is worth while pointing out, however, that nearly all of the new methods of obtaining a better knowledge of abnormalities of anatomy and physiology are, so to speak, impersonal. In fact it is entirely possible, if not commendable, for one at the present time to arrive at an accurate estimate of certain surgical diseases and apply suitable operative treatment without making use of any information conventionally known as the history and physical examination. There is, therefore, often a great expenditure of effort on the part of the young surgeon in training to collect information concerning conditions identical with or different from the standard John Doe without any attempt to get acquainted with John Thompson, III, Michael O'Flaherty, Johann von Ribbentrop, Vittoria Angelucci or Abie Cohen, not to mention the patient being just one individual of one of these groups.

During the days of diagnosis by history and physical examination alone, there was certainly a great deal more opportunity for the young surgeon to find out something about the patient's social position, ambitions, anxieties and other intangibles which, after all, are the only determinant of an individual; and, perhaps even more important, for the patient by revealing his intimate personal attributes to obtain a conviction of confidence in the surgeon, which not only insures a diminished risk of operation, but which, if enduring, contributes greatly to the patient's ultimate restoration to health and happiness.

This matter of a surgeon's obtaining a conviction of confidence in him on the part of the patient is of much more importance than the attention it has received. In fact, I believe I am correct in saying it has been more often discouraged than encouraged. I am quite sure the "success" apparently obtained on the basis of personality rather than ability has been too frequently the subject of ridicule. Inability on the part of an able young surgeon to acquire patients not only contributes to the period of time which he has to wait before beginning to make his contribution to the world, but what is more pathetic than a well trained surgeon without anything to do? The fact that some practitioners of surgery obtain a large clientele by pomposity, the wearing of loud clothes, or advertising just within the rim of acceptable ethics, should encourage rather than discourage the stimulation of well trained young surgeons to acquire an understanding of the psychology of transference.

This brings us directly to the consideration of another special field of interest in medicine—psychiatry—of which I can make no claim of possessing any more knowledge than would be acquired from reading and from 30 years of experience in the practice of surgery in a clinic, the primary purpose of which is undergraduate teaching of medicine and postgraduate training in surgery. This experience has afforded me an excellent opportunity of realizing the close personal relationship which exists between the surgeon and his patient in a large variety of individuals. In this experience I have also been able to enjoy a very close personal relationship with a considerable number of surgeons in the various stages of their development. Each succeeding year I am more impressed with the large number of physical and chemical tests with which the resident surgeon must be familiar, and, I may add, must be able to interpret to his Chief, so that I can condone rather than condemn his viewing an individual patient only from the viewpoint of a mechanism gone wrong. I know that age and experience will so alter his methods of the practice of surgery so that, although he may not admit it, he will subsequently include in his consideration of a sick man many intangibles not revealed in physical and laboratory diagnostic procedures.

My personal relationship to psychiatry has been very well expressed by Dr. Louis Hamman in a paper delivered before the American Association for the Advancement of Science, of which the following is a quotation:

"When I was a medical student, psychoneurotics were objects of ridicule and contumely, not of serious study and sympathy. The patient with no gross lesion to justify his many complaints was neglected and avoided. The worst insult that could be cast at a fellow student was to call him a neurasthenic. After careful observation had established the fact that a patient had no organic disease, professional relations were supposed to be at an end, and he was dismissed with some such reassuring remark as, 'There is nothing whatsoever wrong with you. Your troubles are imaginary. Go on and forget them,' or even more curtly with a placebo and the ardent hope that he would never return.

"With the passage of years this brusque attitude has become somewhat

softened and yet essentially it is still the attitude of many physicians. The mind always seeks precise classification and is never more pleased than when experience can be snugly labeled. Packing it away in this neat fashion is so much more satisfactory than delving about for explanations that require long and painstaking investigation and may in the end elude our search. The great number of elaborate tests now available for diagnosis plays smoothly into this indolent habit of mind. Surely one of them will sooner or later reveal some abnormality, and upon this slender thread of evidence the patient's symptoms conveniently may be strung."

From a present day evaluation of the methods employed by all practitioners of medicine before a comparatively recent period, one is forced to conclude that physicians must have attained recognition for rendering useful service because of their abilities to alleviate human suffering by psychotherapy rather than because of specific materialistic therapy. It is interesting to point out that it is possible that the violence of the therapeutic methods employed by eminent practitioners of the past, such as bloodletting and purgation, were after all the means by which a psychotherapeutic result was obtained. Without any thought of commending the use of the surgical operation as a psychotherapeutic method, it is well for the surgeon to bear in mind that the operation may produce an effect beyond a mere alteration in anatomy or mechanistic physiology.

When psychiatry was recognized as a special field of medicine, the pure psychiatrist was for the most part concerned with the major psychoses, which, after all, were only labeled by the application of descriptive names, and the sufferers were committed to institutions of unbelievably inhuman environment. Psychiatry retained a slender connection with objective medicine through the frequency of its combination in practice with organic neurology, which latter interest has at present almost completely passed into the hands of the neurologic surgeon. The utter pity and loathing of all men for the insane lives today in the almost universal prejudice which even men of medicine hold for a psychiatric consultation, just as the fear and dread of a surgical operation, because of the horrible years before anesthesia and antiseptics, has but recently been greatly reduced.

Even if I were competent to do so, it would take us too far afield to attempt a résumé of the history of psychiatry. It is not necessary for one to do more than attempt to read but a relatively small part of psychiatric literature to find out that a large number of so-called psychiatrists are unable to write papers or books in language which is intelligible, even if the reader makes full use of the most comprehensive dictionary. A recent editorial in the American Medical Journal cites a specific example.

From the psychiatric literature which is intelligible, it is clear that the differences in viewpoint of psychiatrists are such that there is even mistrust in each other. There are the psychiatrists who apparently have completely abandoned all consideration of the somatic aspects of disease, except for a sort of a vague retention of a concept of the frontal cerebral lobes. This group

accepts the psychology which William Morton Wheeler compares to an anatomy based purely on observations of the more aesthetic aspects of the head and upper extremities. Some psychiatrists prefer to consider the penis as the root of all evil. And here it is a case of apparent identity of *have* and *have not*. The psychoneuroses of the male start at the time of discovery of the organ, and those in the female with the discovery of its absence. This school of psychiatry by accepting the existence of an influential unconscious mind, is based on a more realistic and understandable psychology which is consistent with the generally accepted principles of evolution in biology. Unfortunately, this school of psychiatry has been hampered in its development by some of its followers placing too much emphasis on sex and assigning too great a value to psychoanalysis as a therapeutic method. There is a universal prejudice against personal revelation of matters of sex, and it has not been proven that the elevation of components of the unconscious into the conscious mind by the long and tedious process of psychoanalysis is the most important method of dispelling the manifestations of a psychoneurosis. However unpleasant the ideas of the psychoanalysts may be, they have undoubtedly made a great contribution to psychiatry, and this contribution will increase as other components of the unconscious mind are given more consideration.

Some psychiatrists are apparently interested in demonstrating that psychic abnormalities are the primary etiologic factor in the production of organic disease, as, for example, duodenal ulcer. From this group come reports of miraculous cures, usually of a single case, and often seemingly for the purpose of revealing the incompetence of the practitioners of surgery. The following is an extreme example based on "hearsay" evidence, but if every word were true, it is still without value:

"A very famous surgeon sent a young woman who was suffering with toxic thyroid to him with the request that he get rid of some of her fears so she could be operated upon, her pulse being so rapid that surgery could not be considered. The psychiatrist analyzed the girl and at the end of three months sent her back to the surgeon minus fears and minus her goiter. The famous surgeon bewailed the loss of his patient, but expressed happiness over the outcome of the case."

Although there still remains a large amount of distrust of psychiatrists in the minds of the objective practitioners of internal medicine, and the even more objective practitioners of surgery, there is certainly developing a better sort of psychiatry, and, even more important, there will almost certainly come out of the experiences of the present war a better understanding of the psychic aspects of disease. Even a casual examination of the reports of the battle casualties in the various military hospitals is convincing evidence that in spite of the presumable rejection for active military service of all men with existing psychoneuroses, many are accepted without adequate reserve of emotional control to withstand the stress and strain of actual warfare.

Primarily because of my feeling that succeeding groups of young men for whom I was assuming the responsibility for their surgical training were

having to acquire such an ever increasing amount and complexity of objective information concerning surgical patients that there was little time or incentive for understanding or appreciating an individual as something more than a machine gone wrong, we undertook what appeared to some of my associates the dangerous experiment of bringing a psychiatrist and psychologist into the surgical wards. This experiment was made possible by the cooperation of Dr. Frank Luton, head of the psychiatric division of the Department of Medicine, Vanderbilt University Medical School, the generosity of the Rockefeller Foundation, and the good fortune of securing the services of Dr. Smiley Blanton, an experienced psychiatrist, and Miss Virginia Kirk, a well trained psychologist of large medical experience.

It also occurred to me that such a project might contribute to the development of psychiatry as well as giving a broader viewpoint to the young surgeon in training.

I believe the experience of the psychiatrist, at least for the most part, is limited to his dealing with individuals in whom apathy, anxiety, or fear is present to a degree quite without the limits of that which can occur in what, for lack of a better term, one must call the average individual. Furthermore, he is deprived of the opportunity of subjecting individuals with whom he comes in contact to conditions peculiarly effective in producing major emotional responses. He sees mental disease only in its later stages, and perhaps only in individuals who have always been without the range of the normal in emotional responses.

The surgical wards offer daily instances of various degrees of anxiety in individuals, male and female, of all ages and states of emotional stability. Furthermore, the exact time at which a major stimulus arousing anxiety is to be applied is often definitely known, and in many instances could be chosen to suit the problem being investigated.

A study of conditions before and after operation by methods known only to the experienced psychologist would undoubtedly advance our knowledge of both psychiatry and surgery. Before operation there would be the patient whose symptoms of a surgical disease were in reality a psychoneurosis, and the patient with anxiety associated with an organic surgical disease or its treatment. After operation there would be the patient restored to full health, some who had but a few months to live, and perhaps an even more important group, those who have to wait some years for the possible or probable recurrence of a malignant disease.

There is always the difference of opinion as to whether the patient with an incurable disease should be told the truth or a falsehood; and if he is told the truth, shall it be done by gradually working up to the bad news, or should he have the worst news first and subsequently be given every encouragement which it is reasonably possible to give? Then, too, there are the two patients who are to have the same operation the same morning—one dreads "going to sleep" and the other cannot bear the idea of being conscious while "under the knife." In fact, the greatest difficulty encountered in this project was to keep

from spreading too thin over the large number of interesting problems of which we were immediately aware.

It is not the purpose of this paper, nor has the investigation reached such a stage, to give a detailed report at this time, but there has already been some evidence of real accomplishment.

First of all, I wish to stress particularly the reaction of the ward patients to submitting themselves to psychiatric investigation. Almost all of the ward patients came directly or only slightly indirectly from the farms in the vicinity of Nashville. There have been no difficulties because of different languages. They come from a people emotionally as well as geographically stable. During the past year all of the ward patients of surgery have been at least exposed to a psychiatrist, and 64 patients have had a complete psychologic study except for psychoanalysis. Only two patients have not been agreeably cooperative. During the same period on the private pavilion there has been universal objection, and in most cases positive refusal to accept a specific request for psychiatric consultation. The explanation for this difference lies in the fact that the psychiatrists were not labeled on the wards—they were not only permitted, but encouraged to pose as members of the surgical staff.

The frequency of the occurrence of the modification of behavior because of anxiety arising from other sources than the surgical organic diseases and their surgical treatment is illustrated by the results of the psychiatric examination of 14 consecutive cases of acute appendicitis operated upon and found to have definite acute inflammatory process in the appendix. Seven of these cases were found to have evidences of anxiety of sufficient gravity to be of clinical significance. In two cases operated upon during the same period in which no organic change was found in the appendix, psychiatric examination disclosed sufficient evidence to justify the conclusion that the symptoms and signs of acute appendicitis which were such that operation was indicated, were in reality manifestations of neuroses.

The patient with symptoms and signs of acute appendicitis sufficiently characteristic to make delay of operation dangerous, and subsequently at operation found to have no demonstrable pathologic change, constitutes a problem to which surgeons have not devoted the attention deserved. The importance of considering the psychic aspects of malignant tumors, disabling deformity, or unsightly disfigurement is such that preoperative preparation of the patient for these results is just as important as is the transfusion of blood or compensation for vitamin deficiency to reduce the risk of operation and promote the healing process in the operation wound.

It has been of particular interest to find out that only a small proportion of patients manifest any signs of great fear or dread of an impending operation. In reality, more frequently patients have exhibited evidence of relief from anxiety because of the anticipation of being so positively and perhaps so quickly cured by a method which may appeal to them also as an atonement for their past and secret sins. This suggests that the psychoneurotic patient who makes much of her previous operation has not in reality been made a psychoneurotic

because of this operation, but that she is merely utilizing this experience as an integral part of her neurotic pattern. It is also not impossible that the patient who states she has been in absolutely perfect health since a cholecystectomy—with the removal of a perfectly normal gallbladder—is contemplating the operation from the crest of a wave of a mild manic-depressive insanity. Recently a patient upon whom I performed a radical removal of the rectum made a trip from a distant city in order that she might let me know from her own lips what a wonderful asset she had in her permanent colostomy. After listening to her for an hour I decided henceforth I would specialize in the performance of colostomies only in individuals with manic-depressive insanity who were about to emerge into the stage of elation.

I confess that I find it difficult to make definite statements of the reaction of the visiting staff, house staff, and undergraduate students to the inclusion of a modicum of psychiatry in the department of surgery. The visiting staff at least have ceased to make any objections, and on a few occasions I have noted at least a glimmer of interest. The house staff have at least been aware of my own interest in a project, the purpose of which is to shorten the time in which they will acquire that indescribable something frequently referred to as "surgical judgment" and heretofore presumably acquired only by long experience. After all, in this matter it is the state of mind which is important. There is certainly no thought of making even one of my surgical house staff into a psychiatrist, but I cannot but believe that they will be better surgeons for having been stimulated to discover before roentgenologic examination that a man complaining of abdominal discomfort, loss of appetite, weight and strength may have these symptoms because his only son is in the Solomon Islands. The subsequent diagnostic tests may then be conducted with more sympathy and the final diagnosis made and the treatment given with a better prospect of success because of this better understanding. God forbid that the roentgenologic examination should ever be omitted.

All of the undergraduate students assigned to the surgical wards attend a discussion of current problems, led by the psychiatrist, once each week. Their interest is demonstrated by the librarian's finding it necessary to place on the reserve shelf several of the recent books on psychosomatic medicine, which, incidentally, has caused me considerable inconvenience in the preparation of this paper.

Analogous to Mark Twain's often repeated observation concerning the weather, practitioners of objective medicine have been content with calling attention to the fact that the many schools of medical quackery exist solely because reputable physicians and surgeons ignore not only psychic abnormalities, but also the psychic aspects of organic disease. The neurotic patient turns to the quack for help when told there is nothing the matter with him, and not infrequently the patient with incurable organic disease makes use of this last ray of hope with at least the quasi approval of the surgeon. The solution of the problem of quackery will not come from the efforts of organized medicine to abolish it by legislation, as surely as it will follow the introduction of a

better psychiatry into surgery and perhaps a little bit more knowledge of organic medicine into psychiatry.

If one wishes to pursue this ideology further in an imaginary world to a complete surgical Utopia, particularly if he were one who has the responsibility to choose as well as to train surgeons, some of whom he hoped would some time attain the distinction of membership in this Society, he would also at least give some thought to the psychic as well as the somatic aspects of the individual selected. I am quite sure that many of you have known men adequately equipped with knowledge and manual dexterity who, in spite of years of effort, have not attained success as surgeons. Even the best surgeons, I feel sure, have some off days, and at times feel that they have committed sins of omission or commission because of a diminished reserve of emotional control. A good surgeon, therefore, must have not only a sound body, but an adequate control of will to maintain a calm and clear mind under conditions of stress and strain which at times may even be too much for his assistants. At considerable personal inconvenience I maintain my office in the Vanderbilt University Hospital immediately adjacent to the operating rooms for the sole reason of having my house staff feel that immediate aid is available if unexpected difficulties are encountered; and I am quite sure I have thus given courage a good many times to conscientious young surgeons to bring to a successful conclusion operative procedures which otherwise would not have been undertaken. Occasionally I have purposely refrained from helping a young man with a good mind and nimble hands to obtain opportunities for surgical training because I believed he was not emotionally constituted for becoming an accomplished surgeon. It is at least not too much to hope that out of the vast experience which is now being obtained from the application of psychological tests for the selection of men for a great variety of special tasks, something may be developed which will be helpful to the young man in determining his fitness for the widely different fields of medicine, before he may have spent the best years of his life preparing for a profession in which he cannot attain the success for which he has hoped and for which he has labored so long.

SYMPOSIUM ON ABDOMINAL SURGERY

INDICATIONS FOR, AND EXPERIENCES WITH, TOTAL GASTRECTOMY*

BASED UPON SEVENTY-THREE CASES OF TOTAL GASTRECTOMY

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IN 1938, eight cases of total gastrectomy were reported from the Lahey Clinic by one of us (F. H. L.),[1] with three postoperative deaths. Since then, 65 additional patients have been submitted to total gastrectomy, with 21 deaths. Of the entire group of 73 patients, there were 24 postoperative deaths, an operative mortality of 33 per cent. During the last two years, total gastrectomy was performed upon 28 patients, with five postoperative deaths (18 per cent), a notable reduction in operative mortality.

Total gastrectomy has opened up a relatively new field in the treatment of gastric malignancy and has now become an accepted surgical procedure, with a reasonable mortality.

We have always felt that when we had had a sufficiently large experience with total gastrectomy, we should review our records and decide whether, in advising this procedure, we could assure patients a reasonable prospect of life and relief of symptoms. It, therefore, now seems important to present our experiences in terms of whether the patient's existence is satisfactory after a total gastrectomy, how long it prolongs life, and the prospects of cure in the lesser grades of malignancy, such as lymphosarcoma and leiomyosarcoma. It also seems desirable to present our accumulated data and convictions concerning the preoperative selection and preparation of patients, method of approach, types of anesthesia, operability, technical considerations, results and postoperative feeding and diet.

SELECTION OF CASES AND METHOD OF APPROACH

The history of the patient with gastric carcinoma appears to offer nothing of value in determining which patients should be submitted to total gastrectomy. Obviously, exploration is not justified in a patient who has a fixed lesion demonstrable by roentgenologic examination and palpable in the epigastrium, with nodules palpable in the liver. Likewise, exploration is not justified in a patient with a Virchow's node or a rectal shelf.

However, those lesions with rigidity of the entire stomach without obstruction, Virchow's nodes, a rectal shelf, fixation of the lesion, metastatic nodules palpable in the liver, or roentgenologic evidence of involvement of other areas, such as the transverse colon or the loops of small intestine,

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should be explored, even if the roentgenogram shows the stomach to be entirely involved by the lesion. The roentgenogram, therefore, is of great value in determining whether the patient may possibly be submitted to total gastrectomy.

We repeatedly have said that there are two distinct types of carcinoma of the stomach. The localized type, which so often proves inoperable, is frequently on the lesser or greater curvature and rapidly metastasizes to the adjacent lymph nodes. In the other type, the linitis plastica or leather bottle stomach type (Fig. 1), the carcinomatous infiltration is intramural

FIG. 1.—Linitis plastica. Roentgenogram showing complete involvement of the stomach by carcinoma.

and spreads through the walls of the stomach (Fig. 2), streaming down between the muscle bundles in the gastric wall. This is the type of carcinoma that lends itself so frequently to total gastrectomy (Fig. 3). We have often been surprised and have been unable to explain why such an extensive gastric carcinoma of the linitis plastica type can exist with little or no visible lymph node involvement when such rapid involvement occurs in the more localized and more common type of malignant lesion.

In addition to the linitis plastica type of carcinoma, lymphosarcoma with its tendency for mural infiltration (Fig. 4) particularly lends itself to total gastrectomy. It produces gastric rigidity of a less striking character than does the linitis plastica type and is likewise without distant extension.

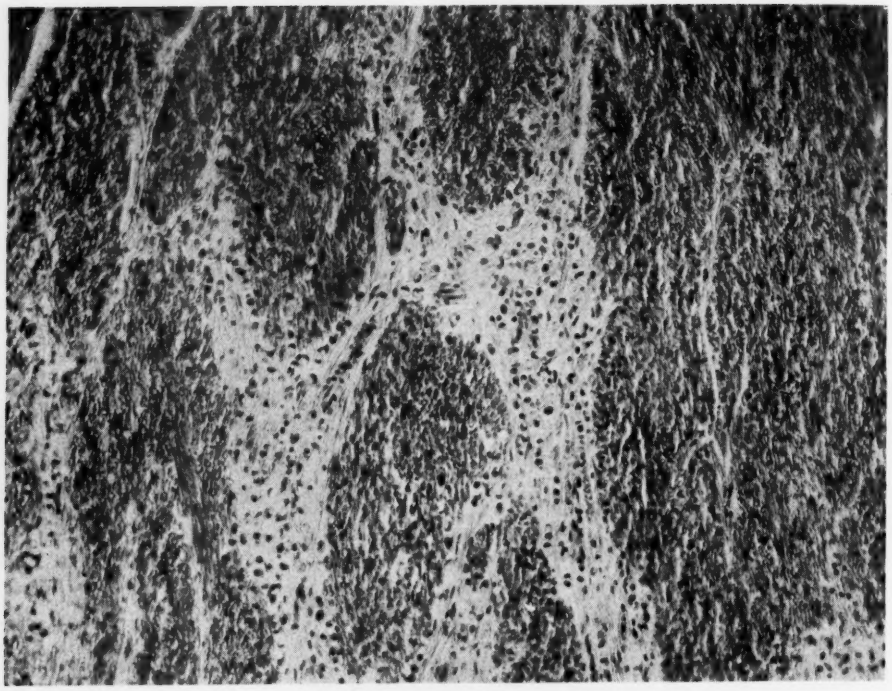

FIG. 2.—Photomicrograph of section of stomach showing carcinomatous infiltration of gastric wall in linitis plastica.

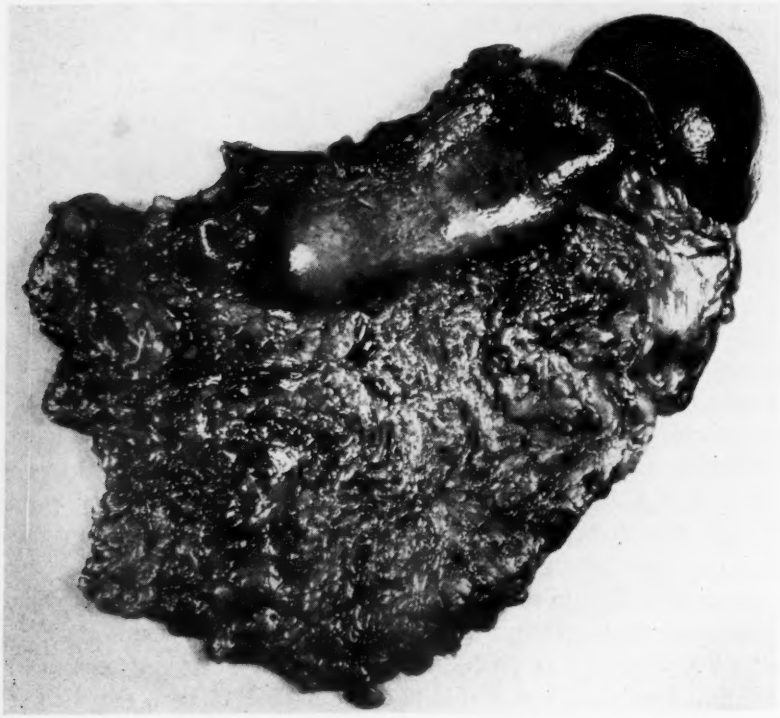

FIG. 3.—Surgical specimen of linitis plastica type of carcinoma after total gastrectomy, splenectomy and omentumectomy.

A third type of malignancy which lends itself favorably to total gastrectomy is the sarcomatous degeneration of multiple leiomyomas (Fig. 5). In this condition the malignancy is of such low-grade character that total gastrectomy offers a real prospect of cure.

A fourth lesion which lends itself favorably to total gastrectomy is carcinoma of the cardiac end of the stomach (Fig. 6). In our group of 73 patients, several had lesions limited to the upper end of the stomach. Previous to the employment of total gastrectomy, these patients were rejected as inoperable. Among these cases are lesions extending up to, but not including,

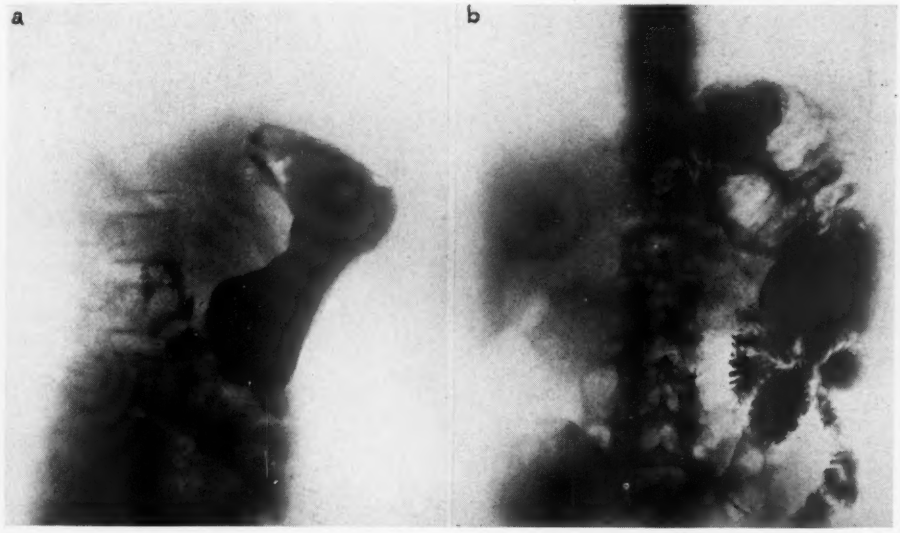

FIG. 4.—Lymphosarcoma. (a) Roentgenogram of stomach before operation showing extensive involvement with lymphosarcoma. (b) Roentgenogram after total gastrectomy showing anastomosis of esophagus to jejunum. Taken one year following operation. Patient in excellent health. Has gained 20 pounds in weight.

the esophagus, and in addition to these and not included in this series, are cases of carcinoma of the stomach resected transpleurally.

From our experience with the two procedures, total gastrectomy with anastomosis of the jejunum to the stump of the esophagus through the abdomen and transpleural resection by means of splitting the diaphragm and pulling the cardiac end of the stomach into the pleural cavity, we believe the former is safer, providing enough normal esophagus remains. Those lesions of the cardia with enough esophagus for anastomosis are better approached through the abdomen with the prospect of employing total gastrectomy, than through the pleural cavity, for the following reasons: With the abdominal approach, a thorough, satisfactory exploration for determination of operability can be performed under direct vision. The pelvis can be examined for gravity metastases and implanted nodules low in the abdominal cavity and enteric lymph nodes can be felt. In addition, removal of the entire omentum and spleen is possible, as well as safe and satisfactory mobilization and inversion of the duodenum. These procedures

FIG. 5.—Leiomyosarcoma. Stomach completely involved by large tumor masses of leiomyosarcoma. Patient alive and well six years following total gastrectomy.

FIG. 6.—Carcinoma of cardia. Roentgenogram showing carcinoma involving cardia of stomach. Patient alive and well four and a half years after total gastrectomy.

necessarily must be limited when the operation is performed transpleurally through the divided diaphragm. For these reasons, as already mentioned, we prefer the abdominal approach to lesions arising in the stomach when there is enough esophagus for anastomosis to the jejunum, and reserve transpleural resection largely for those lesions arising within the esophagus.

PREOPERATIVE PREPARATION

Many patients who must be submitted to total gastrectomy are elderly and invariably represent an increased risk group. They are often anemic and frequently are in a state of poor nutritional balance. The restoration of water and mineral balance is frequently necessary. Blood transfusions often must be given to restore hemoglobin and blood proteins to normal levels. Most of them should have glucose solutions intravenously for liver storage, and vitamin C in sufficient dosage.

For a number of years we have thoroughly lavaged the stomach of all patients with gastric carcinoma the evening and morning before the operation, using a dilute solution of hydrochloric acid (7.5 cc. of concentrated hydrochloric acid in one liter of water). This procedure, which mechanically cleanses the stomach and cuts bacterial counts in the gastric contents down to a minimum, is of value in diminishing infection not only in total gastrectomy but in partial resection for carcinoma.

ANESTHESIA

Usually total gastrectomy can be performed in from one and a half to two hours, depending on the technical difficulties encountered. However, since the length of time necessary is largely unpredictable, all gastric resections, total and subtotal, in this Clinic are now done under fractional spinal anesthesia. By this method, small repeated doses of pontocaine can be given as often as necessary with certainty of complete relaxation during the most difficult technical portion of the procedure, that is, during anastomosis of the jejunum to the stump of the esophagus high up under the diaphragm. Although fractional spinal anesthesia has now been employed for several years with complete satisfaction in all our gastrectomies, we express the warning that it should be administered only by those trained and experienced in its use.

A part of the anesthetist's duty is the introduction of a Levine tube into the stomach. No gastric resection, either total or subtotal, is undertaken in this Clinic without the use of a Levine tube, by means of which it is possible to empty the stomach of gas or fluid. As the completely mobilized stomach is held up (Fig. 11), any fluid within it tends to gravitate into the esophagus, from which it can be evacuated by means of suction applied to the indwelling Levine tube, and consequently it will not spill out into the peritoneal cavity when the esophagus is opened.

After the jejunum is tacked to the esophagus (Fig. 13), the Levine

volving cardia of stomach. Patient alive and well four and a half years after total gastrectomy.

FIG. 5.—Leiomyosarcoma. Stomach completely involved by large tumor masses of leiomyosarcoma. Patient alive and well six years following total gastrectomy.

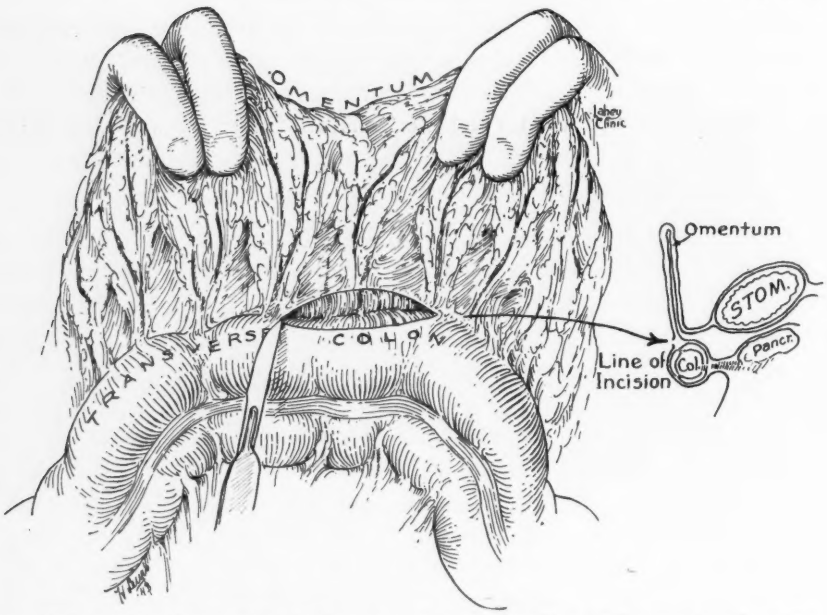

FIG. 7.—The first step in the separation is sharp dissection of the omentum from the transverse colon. Note the avascular line of cleavage. Note the developmental line of cleavage and the line of incision in the avascular area (insert).

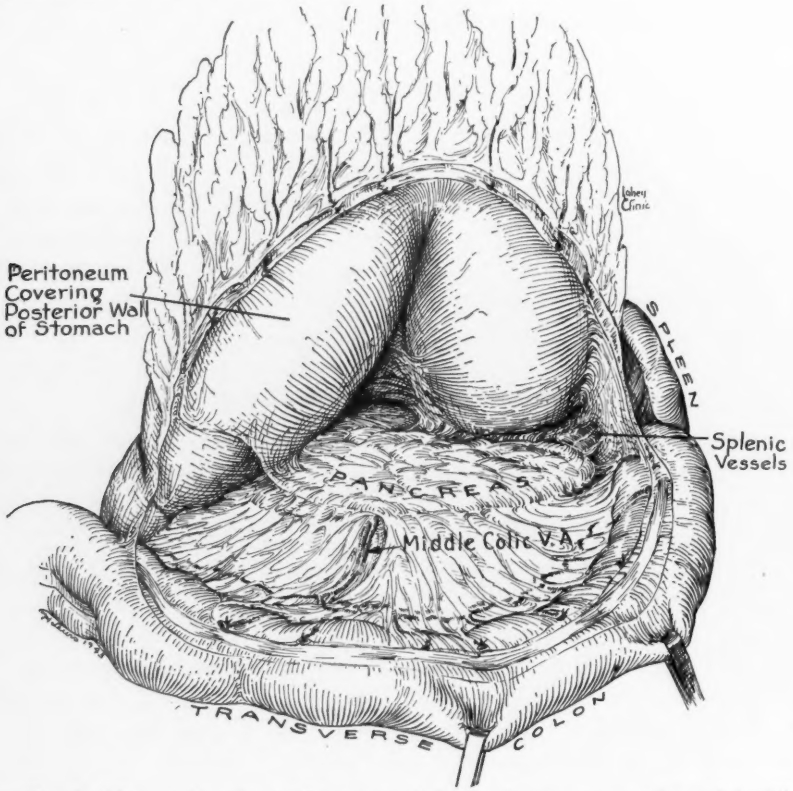

FIG. 8.—The omentum is completely separated from the transverse colon and is still attached to the greater curvature. The pancreas is visualized, and the stomach is turned up. Note the splenic vessels beyond the tip of the pancreas.

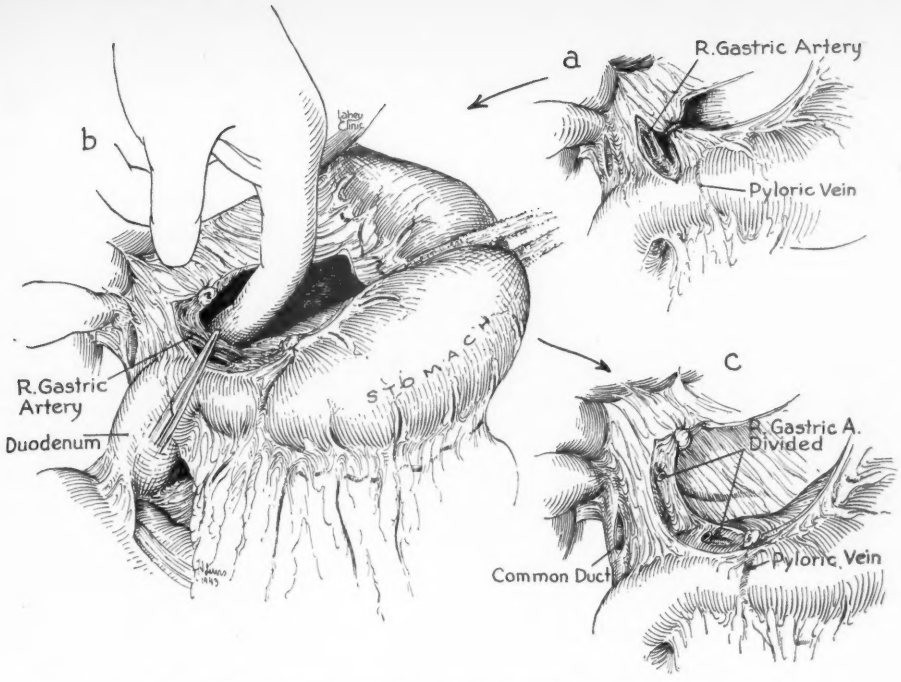

FIG. 9.—(a) The lesser peritoneal cavity is opened through the gastrohepatic omentum.
(b) The right gastric artery and vein are clamped close to the pylorus.
(c) Vessels of the gastrohepatic omentum have been ligated and the lesser curvature is cleared.

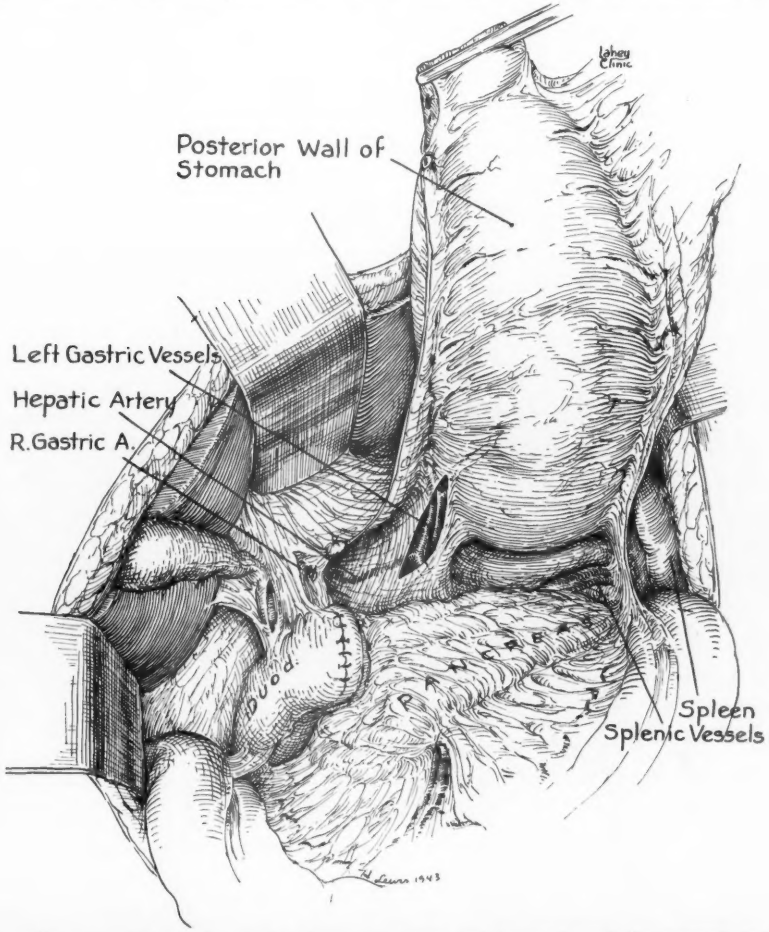

FIG. 10.—The duodenum has been cut across and inverted, and the stomach has been lifted higher. Note the exposure of the left gastric vessels ready for ligation.

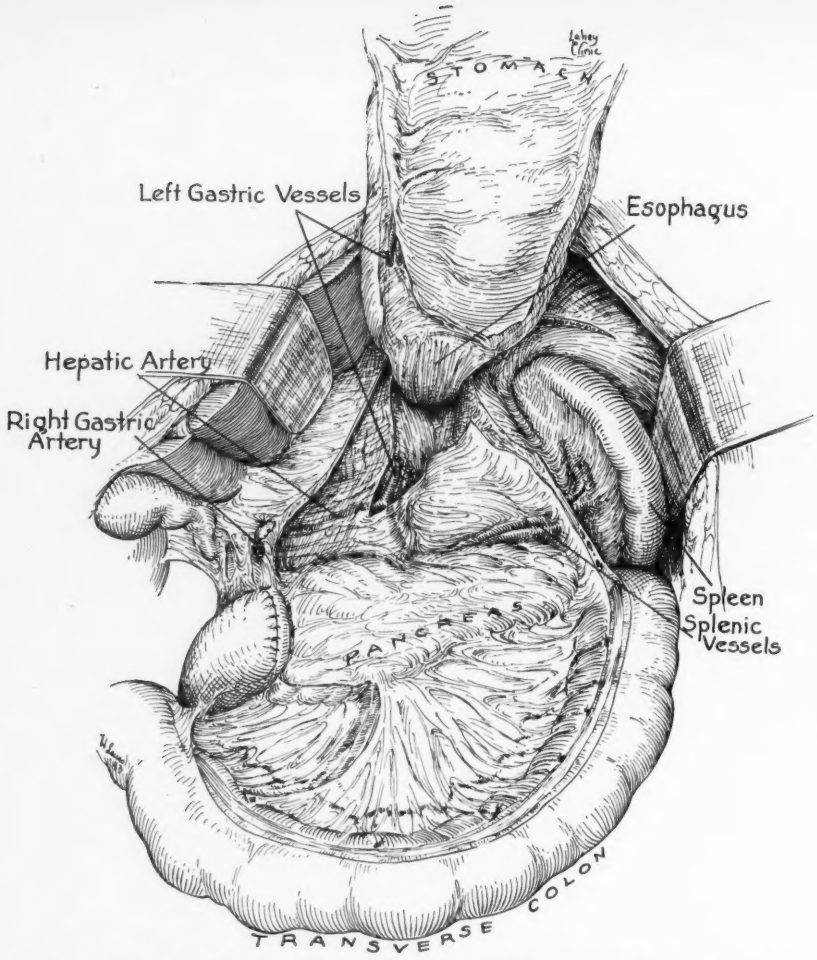

FIG. 11.—The esophagus has been exposed and freed, the stomach has been turned up, and the pedicle of the spleen has been visualized. The left gastric vessels have been ligated. The spleen is ready to be removed with the stomach.

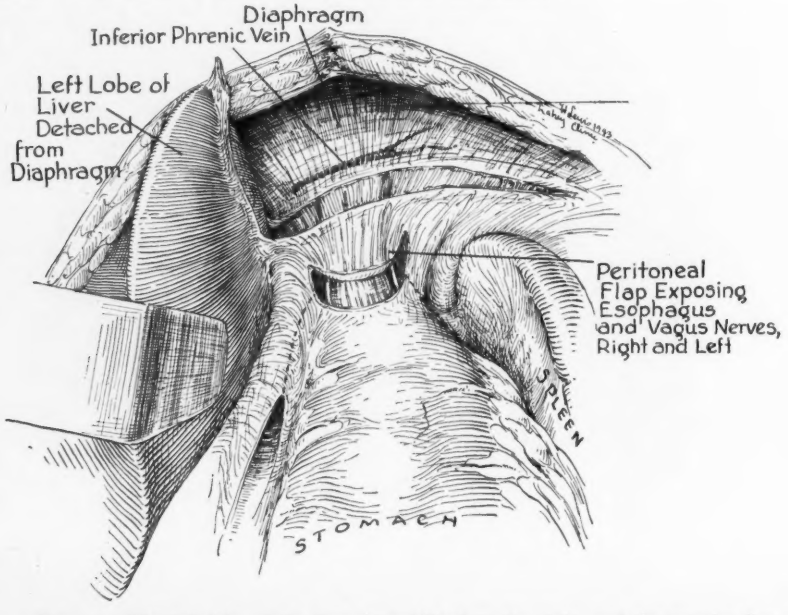

FIG. 12.—The left lobe of the liver is mobilized to the right, and the anterior flap of the peritoneum is reflected up over the esophagus. This is used later to suspend the jejunum in order to remove traction from the anastomotic line.

tube is withdrawn into the esophagus; thereby any accumulation of mucus within that structure can be withdrawn and prevented from passing out of the wound into the operative field. This procedure is extremely important in the prevention of soiling by leakage from the stomach or from the esophagus.

OPERABILITY

All cases considered for total gastrectomy are borderline in terms of operability, although in many cases the decision can be made before operation is undertaken. In others, exploration is justifiable, but the final decision of whether total gastrectomy should be undertaken must be made with the abdomen open. If the operation is to be logically applied, this decision must be extremely critical in character, since total gastrectomy with its relatively high mortality should not be undertaken when there is little prospect of greatly prolonging the patient's life or adding to his comfort. An unwise decision gives the operation a bad name, does not add luster to the reputation of surgery, and results in unnecessary expense and discomfort to the patient.

With increasing experience, we have become more expert in rejecting the hopeless cases. The surgeon should first explore the pelvis to eliminate gravity metastasis. If metastasis is present, regardless of how removable the lesion apparently is in terms of total gastrectomy, operation is, in our opinion, not justifiable. The transverse colon should be turned up and the jejunal fossa carefully investigated for firm nodes or direct extension of peritoneal implants. Since most gastric carcinomas which are favorable for total gastrectomy are of the linitis plastica type and are not ulcerative, firm nodes are generally assumed to be metastatic in character, and again in terms of total gastrectomy, the condition is inoperable. If on examination of the omentum, there are only a few firm nodes, the patient should not be rejected as a candidate for total gastrectomy, since the entire omentum with its nodes can safely be removed with the stomach. When, however, there is direct extension to the root of the mesentery of the transverse colon, the lesion is inoperable. Likewise, when there are peritoneal implants about the stomach, the lesion is inoperable. The surgeon should be particularly careful to palpate for a small nodular chain of lymph nodes which frequently run up through the diaphragm beside the esophagus. These are at times involved with a firm, continuous, shot-like chain of metastatic nodes, and although the lesion is removable, total gastrectomy is not justifiable. Obviously, investigation of the gastrohepatic omentum and liver would not be neglected. Not infrequently, the type of carcinoma one sees in linitis plastica produces thick, white infiltration along the greater curvature, particularly where the spleen is connected to it by the vasa brevia vessels. In these cases removal of the spleen (now routinely done in total gastrectomy) together with the stomach is desirable in order to get well outside the local extension of the malignancy. One of the most important decisions that must be made before attempting total gastrectomy is whether an adequate amount of esophagus remains uninvolved by the carcinoma for a satisfactory

anastomosis between the esophagus and the jejunum. Even with involvement of a small portion of the esophagus, as soon as the parietal and diaphragmatic peritoneum and the vagus nerves are cut, a considerable length of esophagus can still be pulled out of the pleural cavity for a safe and comfortable anastomosis.

TECHNICAL CONSIDERATIONS

Total gastrectomy has come into use so recently that every surgeon who performs the operation has gradually developed technical improvements which make it possible for him to undertake the procedure more expeditiously and more safely.

One of the most important technical features, in our experience, is the separation of the omentum from the transverse colon (Fig. 7), opening the lesser peritoneal cavity, exposing the splenic vein and artery so that they readily can be ligated at the tail of the pancreas, so that the spleen can be turned up and removed with the stomach. Splenectomy, as proposed by one of us (F. H. L.), not only has not added to our mortality, but by improving exposure, may have diminished the number of deaths in the last few years when it has been an almost routine part of total gastrectomy. The omentum should be entirely freed from the transverse colon well over on the right side and separated well up to and including its attachments at the splenic flexure (Fig. 8). After ligation of the splenic vessels, the mobilized spleen is attached to the stomach only by its vasa brevia vessels. With the stomach turned up, it is important to control the blood supply of the stomach from behind, with the lesser peritoneal cavity open and with the pancreas visualized (Fig. 10). The left gastric artery can now be ligated high under direct vision, and all bleeding vessels on the lesser and greater curvatures of the duodenum can be controlled (Fig. 9). The duodenum is clamped across and closed by open inversion with three rows of sutures, the last one being silk, and is then covered with the capsule of the pancreas. The omentum, spleen and stomach in one mass can now be wrapped up and tied in a large pad. The esophagus can be wiped free of the areolar tissue and pulled down as fas as possible out of the chest. One of us in an earlier communication (F. H. L.)[1] suggested at this stage the detachment of an anterior and posterior peritoneal flap from that portion of the diaphragmatic peritoneum reflected over the esophagus (Fig. 12), to be utilized later to suspend the jejunal anastomosis to the esophagus, thus taking dangerous traction off the line of anastomosis (Fig. 15, c). This is extremely important because the esophagus is friable and at the best holds sutures poorly.

Anyone who has dealt much with total gastrectomy has been impressed with the fact that the vagus nerve on each side of the esophagus as it passes through the diaphragm greatly limits delivery of the esophagus. With the esophagus clearly exposed, both vagus nerves can be seen, and when severed immediately permit a surprising section of the esophagus to be pulled down from within the pleural cavity, thus bringing the esophagus closer to the

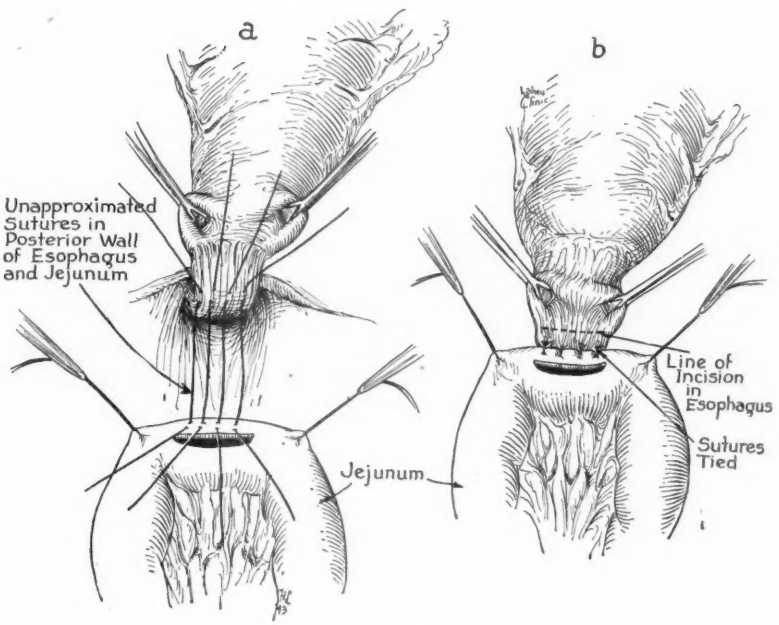

FIG. 13.—(a) The preliminary introduction of a posterior row of untied silk sutures before opening the jejunum. Note the ease with which the jejunum may be opened and trimmed to fit the esophagus. (b) When the sutures are put in and tied before the jejunum is opened, satisfactory management as to size and fit of the jejunal opening is difficult with the jejunum pulled high up under the stomach.

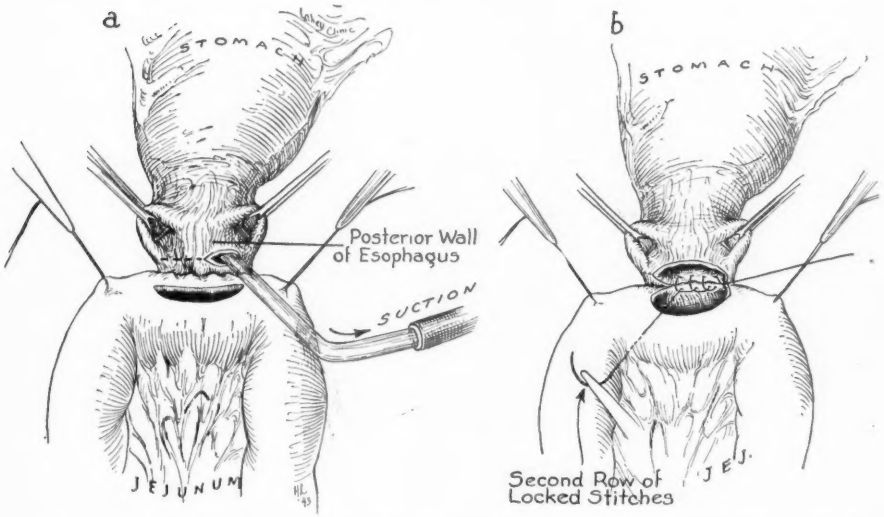

FIG. 14.—(a) The traction stitches to hold the anastomotic opening are spread apart. The first row of silk stitches already has been tied. Note the small opening in the esophagus. (b) Note the locked stitches employed a step at a time as the opening gradually is increased in size until the entire posterior second row of stitches has been inserted.

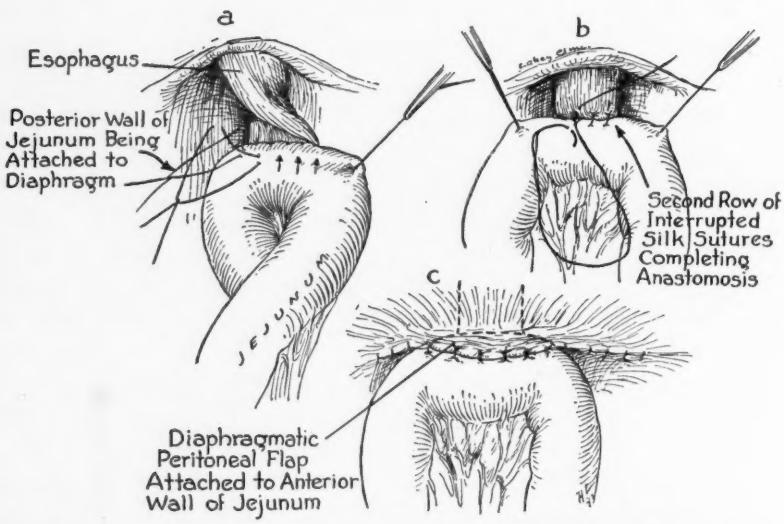

FIG. 15.—(a) The method of twisting the esophagojejunal anastomosis in order to inspect the posterior row of sutures, and the insertion of suspensory jejunal sutures posteriorly to take the strain off the posterior row of the anastomotic suture line. (b) The application of the anterior second row of silk approximation sutures. (c) The previously fashioned flap of diaphragmatic peritoneum sutured to the jejunum over the anastomosis to reinforce it, to suspend the jejunum and to relieve traction on the anastomotic line.

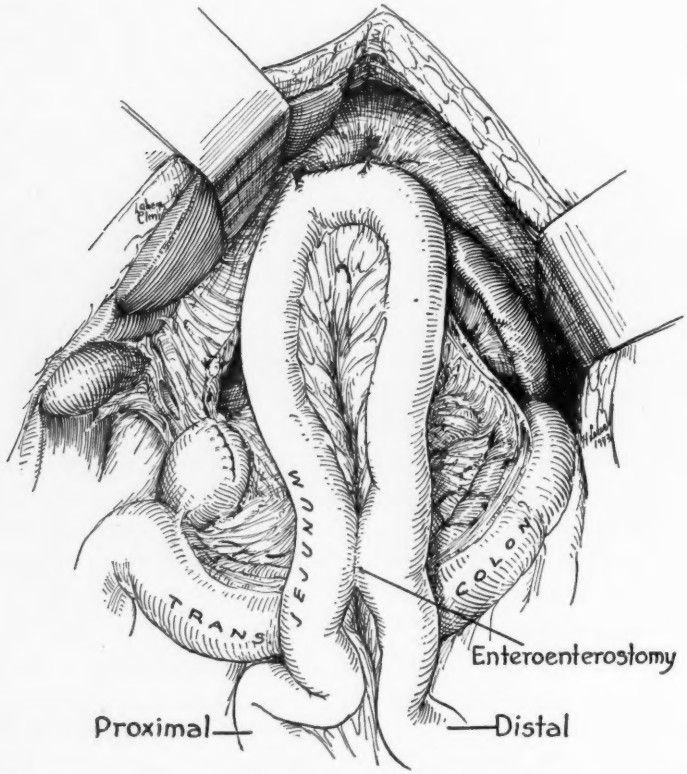

FIG. 16.—The antecolic esophagojejunal anastomosis with additional suspension sutures to the diaphragm. Note the entero-enterostomy.

abdominal wall where the sutures can be introduced more safely and satisfactorily.

One of us (S. F. M.) has employed the plan of placing a posterior row of untied silk sutures before the intestine is approximated to the esophagus (Fig. 13). This facilitates incision of the jejunum and permits trimming of the mucosa of the jejunum down so that it will fit the esophagus. The opened jejunum can then be slipped up against the esophagus as the sutures are gently tied. This procedure has a great additional advantage of permitting the esophagus to be held wide open by stay-stitches during anastomosis (Fig. 14).

In our early cases, postoperative dilatation occasionally was required because of narrowing of the anastomosis between the esophagus and jejunum. In one patient, shown by autopsy to have had no recurrence, we feel sure death could have been avoided had the family physician sent him in for a dilatation when late narrowing of the anastomotic opening occurred, and starvation resulted.

Entero-enterostomy, which has been employed by one of us (S. F. M.), has the advantage of relieving any pressure on the suture line where, as the result of swelling at the anastomosis, there is interference with the passage of fluid from the proximal to the distal loop (Fig. 16). In addition, it has the advantage of side-tracking a considerable portion of the irritating bile and jejunal content into the distal loop and avoiding esophagitis which occasionally results. We will, we believe, now make it a routine part of total gastrectomy.

Anastomosis between the jejunum and the cut end of the esophagus is impossible without some soiling from esophageal content. Therefore, it is advisable to introduce 4 or 5 Gm. of sulfanilamide under the diaphragm about the esophagus to protect against infection. Drainage has not been employed.

RESULTS

As stated before, 24 patients in this group of 73 patients failed to survive operation, a total operative mortality of 33 per cent (Table I). But of the 28 patients submitted to total gastrectomy in the last two years only five have died, a mortality for that period of 18 per cent.

TABLE I

CAUSES OF DEATH IN 24 PATIENTS WHO FAILED TO SURVIVE OPERATION

Peritonitis	14
Shock	2
Coronary occlusion	2
Myocardial failure	2
Pulmonary embolism	2
Bronchopneumonia	1
Mediastinitis	1
Total	24

As would be expected, most deaths were due to intra-abdominal infection. Because of age and grave nutritional changes, these patients obviously are a poor risk group and withstand peritoneal contamination poorly. The rarity of serious pulmonary complications is gratifying, only one patient in this group having died of pneumonia. This is due to better anesthesia and to early recognition and treatment of pulmonary atelectasis by suction bronchoscopy. There were two deaths from shock which undoubtedly represent, in our early experience, too much surgery on extremely poor risk patients.

One would expect mediastinitis to be a more frequent complication in view of the fact that the route to the mediastinum is opened by dissection about the esophagus. However, prevention of soiling, together with careful sealing off of the mediastinal region by suturing the peritoneal flaps of the diaphragm to the esophagojejunal anastomosis, appears to prevent this serious complication.

With added technical and selective experience, the operative mortality following total resection definitely has diminished. Factors affecting this decrease have been discussed, but the fact remains that the prognosis depends largely upon whether the tumor is limited to the stomach or already has invaded the lymph nodes. Most malignant conditions of the stomach are diagnosed late when there is extensive involvement of gastric tissue with invaded adjacent lymph nodes, even though they may not be macroscopically apparent. It is in these cases that we commonly employ total gastrectomy. The prognosis then depends upon the pathology of the growth and the absence of distant metastases. Even though gross involvement of adjacent lymph nodes may not be apparent at operation, in practically all our cases lymph node invasion was noted upon microscopic study. Obviously, most of the resected malignant lesions were carcinomatous, and 87 per cent showed lymph node invasion. Whether the neoplasm was carcinoma simplex or adenocarcinoma seemed to make little difference in prolongation of life. More favorable results were obtained in those patients whose tumors were lymphoblastomatous or leiomyosarcomatous in type. There were five lymphoblastomas and two leiomyosarcomas.

With such a high percentage of lymph node involvement, the opportunity for cure is limited. However, even with recurrence the opportunity to prolong life is considerable. In our own experience with explored and nonresected carcinoma of the stomach, the average life expectancy is five months. If we can expect a period of reasonable comfort for the patient and if the surgical hazard is not too great, total gastrectomy is justified.

In addition, there is always the possibility of cure, as in the case of a young woman now alive and well, who had a total resection six years ago for a leiomyosarcoma involving the entire stomach and previously closed as inoperable. It is well to emphasize that although recurrence will occur in the majority of cases and prove fatal, these patients enjoy freedom from marked gastric distress and when recurrence does take place, it commonly appears

in the liver, peritoneum and retroperitoneal lymph nodes and very rarely involves the esophagojejunal anastomosis.

TABLE II

SURVIVAL PERIOD IN 23 PATIENTS WHO DIED OF RECURRENT MALIGNANCY

Less than 6 months	3
6 months to 1 year	6
1 year to 18 months	6
18 months to 2 years	6
2 to 3 years	1
3 to 4 years	1
Total	23

Table II shows 23 patients who survived operation and subsequently died of recurrence of their malignancy. Of these 23, only three failed to survive less than six months. Therefore, only three of 23 patients who ultimately died of recurrence lived a shorter period of time than they would have if they had not undergone surgery. Fourteen of the 23 lived one year or longer, one of which lived over two years and another over three and a half years. Furthermore, the death of these patients from recurrence was not more distressing than in the unoperated group, and in the majority was even less so. In most of these patients, death was due to cachexia or terminal bronchopneumonia.

TABLE III

SURVIVAL PERIOD OF 26 PATIENTS WITH NO RECURRENCE FOLLOWING TOTAL GASTRECTOMY

Less than 6 months	8
6 months to 1 year	6
1 year to 18 months	4
18 months to 2 years	3
2 to 3 years	1
3 to 4 years	1
4 to 5 years	1
5 years plus	2
Total	26

Of the 73 patients undergoing total gastrectomy, 26 patients are alive and as yet present no definite evidence of recurrence (Table III). Eight were operated upon too recently (less than six months ago) to evaluate the results, and six others have survived for six months to a year. Twelve patients, or nearly half of those alive, have lived a year or longer since operation.

All of these patients are seen at intervals and apparently are in reasonably good health. They are able to take sufficient nourishment to maintain their weight and in some cases even gain in weight. After several months, most patients have been able to carry on their usual activities and employment. One woman, age 35, has been alive and well for four and a half years. A man, age 50, has survived operation for four and a half years after total

gastrectomy for adenocarcinoma of the cardia. A woman, age 33, is well six years after total gastrectomy for leiomyosarcoma.

Late postoperative obstruction of the lower end of the esophagus at the site of the anastomosis with the jejunum may occur. Symptoms of esophageal obstruction do not necessarily mean recurrence of tumor, and in the majority of cases are due to scar tissue narrowing which may easily be corrected by dilatation. We have emphasized the importance of avoiding any narrowing of the esophagus at this point during suturing of the jejunum to the end of the esophagus. If difficulty in swallowing as the result of scar tissue contraction of the stomachal orifice should develop, it may be corrected by simple dilatation with an olive-tipped bougie passed over a swallowed string. Fluoroscopy will disclose the source of the dysphagia, and if necessary, direct esophagoscopy can be employed to rule out recurrent malignancy.

POSTOPERATIVE FEEDING AND DIET

The feeding of patients after complete extirpation of the stomach is divided into stages. In the first stage, the main objective is to supply the patient with some fluid and nutrition, without putting any strain on the suture line. In the second stage, the objective is to establish the patient on a feeding program which will supply him with sufficient calories and other dietary essentials and at the same time prevent the occurrence of certain symptoms which are largely mechanical and due to the loss of the stomach.

For the first three days postoperatively, nothing is given by mouth and fluids are supplied parenterally. On the fourth day, 1 oz. of water is given by mouth every hour. On the fifth day, 1 oz. of malted milk or gruel, made with water, is added at hourly intervals so that the patient gets 1 oz. of fluid every half hour, alternating water and malted milk. On the sixth and seventh days, these quantities are increased 1 oz. each day; and on the eighth day, a single feeding of soft, solid food, such as custard, soft egg, or cooked cereal is added. From this point on, daily increases in the food intake usually are well tolerated; but for the first 15 days postoperatively, only soft solids and liquid foods are permitted.

Supplementary vitamins should be supplied by giving the patient orange juice diluted with water, half and half, in 3 oz. quantities in place of the plain water, at least three times a day. Parenteral injections of vitamin B complex in 1 cc. doses should be given daily beginning immediately after the operation, and continued for about ten days, when the oral administration of vitamin B complex can be substituted.

The second period of dietary management begins about 15 days postoperatively, when the permanent maintenance program is started. A liberal and varied diet should be prescribed, including meat and vegetables, bread and fruit, with the following consideration: The jejunum, which is anastomosed directly to the esophagus, should be protected against trauma from the swallowing of rough or coarse food. If the patient cannot chew his food well, the meat should be ground. Only cooked fruit and vegetables should be

given, and if the vegetables are cooked to the point of tenderness, they need not be puréed. Hard, dry crackers or toast should be avoided. The patient should be instructed to eat slowly.

The absence of the stomach at first permits the patient to eat only small quantities of food at a time. For this reason, it is necessary that only small amounts of food be given, but at least six feedings a day are necessary to supply a sufficient caloric intake. He is eventually able to get along on three to four meals a day.

Some patients complain of fullness, weakness and sweating immediately after the ingestion of food. This symptom appears to be due to the rapid distention of the small intestine. Usually eating more slowly, taking less fluid with the meal and using mainly solid or soft solid foods will control this symptom.

In spite of the loss of all the gastric tissue, a primary type of anemia has not been encountered after total gastrectomy. Postoperative secondary anemia is a frequent complication to be watched for, and when it does occur, the patient should be put on a regular maintenance dose of liver extract parenterally.

SUMMARY

The operative mortality of total gastrectomy definitely has diminished. Twenty-eight operations have been performed in the last two years, with five deaths, a mortality of 18 per cent. Seventy-three cases are reported, with 24 deaths. The operative mortality for the whole group is 33 per cent.

The indications for total gastrectomy are discussed, and the careful selection of patients whose lesion is suitable for total gastrectomy is emphasized. Care must be exercised to avoid resection in hopeless cases.

Technical methods which we have found valuable in performing this operation are described and illustrated.

Prolongation of life may be expected in a large percentage of patients, but few if any cures will result from total gastrectomy except possibly in the leiomyosarcomas.

Out of 48 patients surviving total gastrectomy, 15 patients lived for a year or longer before recurrence caused their death. Twelve patients are alive and well, having survived one year or longer. Seven have lived two or more years. One patient is alive and well after four and a half years, and another is quite well six years after total gastrectomy.

REFERENCE

[1] Lahey, F. H.: Complete Removal of the Stomach for Malignancy: Report of Five Surgically Successful Cases. Surg., Gynec., & Obst., **67**, 212–223, August, 1938.

DISCUSSION.—DR. J. SHELTON HORSLEY, Richmond, Va.: Doctor Lahey has been one of the pioneers who has helped to put this operation on the map. His suggestions in technic are extremely helpful. I think one of the most important is showing that the spleen can be taken out with impunity, because if it is close to the stomach, tacking it up is quite inadequate in many cases.

Total gastrectomy, while not as rare an operation as it was a few years ago, is by no means common now. The indications for such an operation are infrequent. The mortality rate up to recent years has been very high and there has been a general impression that removing all of the stomach would interfere with the hemopoietic process and produce pernicious anemia. The mortality rate has decreased in recent years, partly due to better selection of cases and proper after-treatment, and partly also because of improvement in technic. Many surgeons have contributed to this improvement. Lahey has shown that removal of the spleen with total gastrectomy is apparently of no great consequence. In many instances the spleen may be injured while performing this operation or may be closely adherent to the stomach. If the spleen is injured, suturing or packing it is often not adequate. Allen has shown that suturing the loop of jejunum to the diaphragm tends to lessen traction on the sutures. But probably the most important addition to the technic is that of Roscoe Graham, who brings down the esophagus by blunt dissection with the fingers, severs the vagus nerves, and sutures the loop of jejunum as high as possible parallel to the esophagus. Then, after dividing the esophagus about an inch lower down, he implants the end of the esophagus into a transverse incision in the jejunum.

One of the most interesting articles on the late results of total gastrectomy is by Farris, Ransom and Coller,* in which they review the work that has been done from a physiologic standpoint and the observations they have made on the total gastrectomies done at their Clinic. These authors state that the anemia resulting from total gastrectomy, or even sometimes from partial gastrectomy, is not the macrocytic or pernicious type, but is distinctly hypochromic microcytic or secondary anemia, which is usually associated with iron deficiency. It is well known now that organic iron in food is not available for absorption unless released by peptic or tryptic ferments. This holds true equally if there is achlorhydria. Ferrous iron (sulphate, chloride or carbonate) seems to be the only form in which iron is satisfactorily absorbed from the alimentary canal. According to these writers, the administration of ferrous sulfate three times a day for one week out of every month prevents this secondary anemia. After a few months the patient is able to take practically any kind of food, though it must be in smaller quantities and at more frequent intervals.

I have performed 16 total gastrectomies. The first was in 1929, and Waltman Walters, who reported a case at that time, said there were only 70 cases on record in the literature. My twelfth case was operated upon in March, 1942. All these patients died following operation, and all the operations were for very extensive cancer except one; that was for multiple ulcers, one of them perforating from the cardiac end of the stomach. Two of the deaths were from pneumonia, one from shock, one from volvulus of the bowel between the entero-enterostomy and the esophagus, and one from thrombosis of the superior mesenteric vessels. The other deaths were from infection, subdiaphragmatic abscess, or general peritonitis. In four cases the sepsis resulted from a leak at the suture line; in the others, sepsis developed apparently independently of a leak.

Postmortem examinations were held in 11 of the 12 cases and some knowledge was gained from most of these. In the patient that died from volvulus of the jejunum between the entero-enterostomy, and the esophagus, there seemed to be an indication for a "three-point landing," uniting the right limb of the jejunal loop to the stump of the duodenum. This not only stabilizes the loop and prevents a volvulus, but gives an adequate exit for the contents of the duodenum. In addition to that, I have been placing a rather large drainage tube into the esophagojejunal anastomosis and suturing it there with chromic catgut. This enables the jejunum to be pulled well up over the anastomosis without fear of causing obstruction. The enterostomy for feeding purposes is made with a mushroom catheter a short distance below the entero-enterostomy. The entero-enterostomy is made

* Farris, J. Matthews, Ransom, Henry K., and Coller, Frederick A.: Total Gastrectomy: Effects upon Nutrition and Hematopoiesis. Surgery, **13**, 823-832 June, 1943.

by the rubber band technic described in the October, 1943, issue of the ANNALS OF SURGERY, and in a paper to be presented by me before the present session of the Southern Surgical Association. The technic is simple and reasonably safe.

It is rather remarkable that I should have had four cases this year, all of them successful from an operative standpoint.

CASE REPORTS

Case 1.—W. B. L., a woman, age 50, was admitted to St. Elizabeth's Hospital May 24, 1943. She gave a history of having had "stomach trouble" for 12 months and had lost 25 pounds in weight. She had had pain four or five hours after eating. There were nausea and vomiting, with relief after vomiting. On admission, gastric analysis showed free hydrochloric acid 10, 30 and 70 in the three half-hour specimens. The blood examination showed Hb. 75 per cent; R.B.C. 4,070,000; W.B.C. 7,650. The operation as described was performed May 28, 1943. It was necessary to remove the spleen. There was involvement of the pancreas near the region of the gastric artery. The adherent portion of the pancreas was excised with the hot electric cautery. She made a very satisfactory recovery; her temperature was never above 99.8° F. except on June 10, when she had an attack of pyelitis. She was discharged from the hospital June 24, 1943. Her family physician, Dr. E. L. Johnson of Bedford, Va., who referred the patient, reported on September 1, 1943, that she was in very good condition and doing much of her housework. A letter received from the patient on September 8 says she has an excellent appetite, can eat almost anything, usually has some discomfort for a short time immediately after meals, and is feeling quite well. This discomfort is doubtless due to distention of the jejunum, and should disappear.

Case 2.—R. T. C., a man, age 64, was admitted to St. Elizabeth's Hospital July 12, 1943. He gave a history of intermittent severe epigastric pain for several years. The pain had been worse during the past eight months, during which time he had lost 40 pounds in weight. For the past two weeks there were repeated gastric hemorrhages. On admission his hemoglobin was 24 per cent, R.B.C. 2,070,000. On the following day he had hemiplegia of the right side. Numerous transfusions were given with only a temporary rise in hemoglobin. Gastric analysis showed no free hydrochloric acid in any of the three half-hour specimens. Roentgenologic examination showed marked gastric retention. The examination was repeated several days later but was not satisfactory as the paralysis prevented manipulation. It was apparent that the patient would bleed to death. On August 3, 22 days after admission, he was operated upon under local anesthesia. As in the previous case, there was slight invasion of the pancreas in the region of the gastric artery. This affected portion of the pancreas was dissected off, remaining attached to the stomach, after clamping and dividing the gastric artery. The cancer was extensive. The operative technic described was used. He made a satisfactory recovery, his temperature was never above 100.5° F., and he left the hospital August 25, 1943, 22 days after operation. His paralysis had somewhat improved and his general condition otherwise was very good. He died November 6, 1943, apparently a cerebral or cardiac death.

Case 3.—W. M., a man, age 60, was admitted to St. Elizabeth's Hospital August 31, 1943, with a history of general weakness, substernal and epigastric discomfort for the past six months, with a loss of 12 pounds in weight during that time. On admission to the hospital, the blood examination showed hemoglobin 48 per cent, R.B.C. 3,450,000; W.B.C. 6,000. Gastric analysis gave no free hydrochloric acid in any of the three half-hour specimens. Roentgenologic examinations revealed a very extensive gastric lesion. He was operated upon September 3, 1943, under ethylene anesthesia, by the technic described. His gallbladder, while not involved in the neoplasm, was very tense and adherent, and was removed. There was extensive cancer along the lesser curvature. He made an uneventful recovery.

Case 4.—R. H. G., a man, age 46, was admitted to St. Elizabeth's Hospital October 19, 1943. He was operated upon October 21, 1943. He had a very extensive perforating ulcer from the cardiac portion of the stomach into the pancreas. There were also three other ulcers near the pyloric end of the stomach. In removing the large lesion, which had as its base the pancreas, it was at first thought that it was malignant, although it could not be well removed without sacrificing so much of the stomach as to make total gastrectomy indicated. The portion of the pancreas that was resected was mostly necrotic. In a few days the patient developed a pancreatitis which blocked the common duct as well as the pancreatic duct, and the icterus index gradually went up from 18, seven days after operation, to 100, 14 days after operation.

The liver was somewhat enlarged. A cholecystostomy was performed, and the gallbladder was found dilated and contained much bile. The patient gradually improved and at present, while still in the hospital, is doing well. The margins of the ulcer showed definite inflammation and epithelial hyperplasia but no malignancy.

After losing 12 consecutive cases of total gastrectomy, the successful outcome in four consecutive cases is somewhat gratifying, but in looking back, it is humiliating to recall that if the technic that I have adopted now had been used then, many of these patients that died might have recovered. The improvement in this technic, aside from the general preparation of the patient and the selection of the cases for operation, consists in the principle of Roscoe Graham in making the esophagojejunal union, the insertion of a large rubber tube in the anastomosis which permits the telescoping of the esophagus into the transverse incision in the jejunum without occluding the lumen of the anastomosis, the uniting of the right limb of the loop of jejunum to the stump of the duodenum, the aseptic entero-enterostomy by the rubber band technic, and the use of sulfanilamide and sulfathiazole in the peritoneal cavity.

DR. ROGER D. DOUGHTY, Columbia, S. C.: I have just one word to say: We are still faced with the problem of early recognition of carcinoma of the stomach. I would like to report the death of a patient nine years following a subtotal resection for early carcinoma, performed by Doctor Finney. In my personal experience, which is very limited, this is the longest survival which has come to my attention.

DR. SAMUEL F. MARSHALL, Boston, Mass. (closing): Doctor Lahey has emphasized the necessity for critical selection of those patients to be submitted to total gastrectomy. Obviously, we must make the final decision after the abdomen is opened. We may suppose from the roentgenologic, and other findings, that it is possible to do a total resection, but our positive decision must be made at the time of abdominal exploration. I think it is important to point out that it is well first to visualize the esophagus and determine whether we have enough esophagus uninvolved to make a safe anastomosis. Furthermore, we must make certain there is no extension of the tumor to the pancreas or dissemination to the lymph nodes. It is perfectly useless to attempt to do a total gastrectomy if we cannot take out all the tumor. It is embarrassing to mobilize the stomach and then find that the tumor extends much further than anticipated.

I think that all our efforts have been toward developing a more simple operation, and toward that end Doctor Lahey has suggested the addition of splenectomy to the procedure. I must confess I was hesitant to adopt this at first because I was afraid of the added mortality, but in going over the data I am certain this fear was not justified. Our mortality has not been increased and the operation has been greatly simplified with splenectomy. We have done 28 total gastrectomies over the last two years and our mortality has dropped to 18 per cent.

There is one other point of significance, and that is, the problem of dysphagia that occurs occasionally after operation. We must attempt to prevent narrowing of the esophagojejunal anastomosis during the operation, but if this does follow operation it can be readily handled and very easily diagnosed by direct esophagoscopy and by fluoroscopy. Therefore, dysphagia is not necessarily evidence of recurrent malignancy, and such a diagnosis should not be made without adequate proof.

We have seen hypochromic anemias of the secondary type and, as Doctor Horsley has pointed out, they have been easy to handle. We have had no anemias similar to primary anemia develop after operation.

ACUTE PERFORATION IN GASTRODUODENAL ULCERATION: WITH SPECIAL REFERENCE TO END-RESULTS*

W. L. Estes, Jr., M.D., and B. A. Bennett, Jr., M.D.

Bethlehem, Pa.

The present World War conflict and all its attendant alteration of the normal in living conditions, environment, and emotional strain has again brought the problem of gastroduodenal ulceration, "peptic ulcer," to the fore. The tendency to reactivation of this prevalent gastro-intestinal lesion that war time conditions provide would seem to make timely a consideration of the outcome and end-results of one of its most serious complications.

An analysis, therefore, has been undertaken of 80 consecutive cases of acute perforated gastroduodenal ulceration which were treated at St. Luke's Hospital between the years 1928 and 1942, inclusive. There were seven postoperative deaths, an operative mortality of 8.7 per cent. Of the 73 cases who were discharged from the hospital, no satisfactory report of the subsequent course was available in ten. There were ten subsequent deaths, leaving a total of 53 cases who have adequate follow-up records for review. While certain statistical data will be briefly reviewed, stress will be laid particularly on end-results of those recovering from peptic ulcer perforation.

I—INCIDENCE

A. *Age.*—The age incidence of patients in this series agrees in general with that of other reports. The greatest number (28.7 per cent) occurred in the fifth decade, 22.5 per cent in the sixth decade, 18.7 per cent in the third decade (Fig. 1). However, the largest number of duodenal perforations (30 per cent) occurred in the third decade; whereas, in the gastric group, the highest incidence was in the fifth and sixth decades (31.2 per cent) (Fig. 2), which might be anticipated inasmuch as the usual onset of *duodenal* ulcer symptoms is before 30 years of age and *gastric* ulcer symptoms, not infrequently until after 40.

B. *Sex.*—Of the 80 patients, only two were females; 78 were males (97.4 per cent)—a ratio of one female to 40 males.

C. *Seasonal.*—In this series, there were 27.5 per cent perforations in the Spring, 27.5 per cent in the Summer, 22.5 per cent in the Fall, and 22.5 per cent in the Winter. There is, therefore, no indication of any definite seasonal variation. This compares rather closely to DeBakey's figures (collected series) which indicated for the Spring, Summer, Fall, and Winter 24.6, 24.2, 27.9 and 23.2 per cent, respectively.

II—ETIOLOGY

In 40 cases, sufficient historical data were obtainable to indicate a possible etiologic factor for the perforation. These may be summarized as follows:

* Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

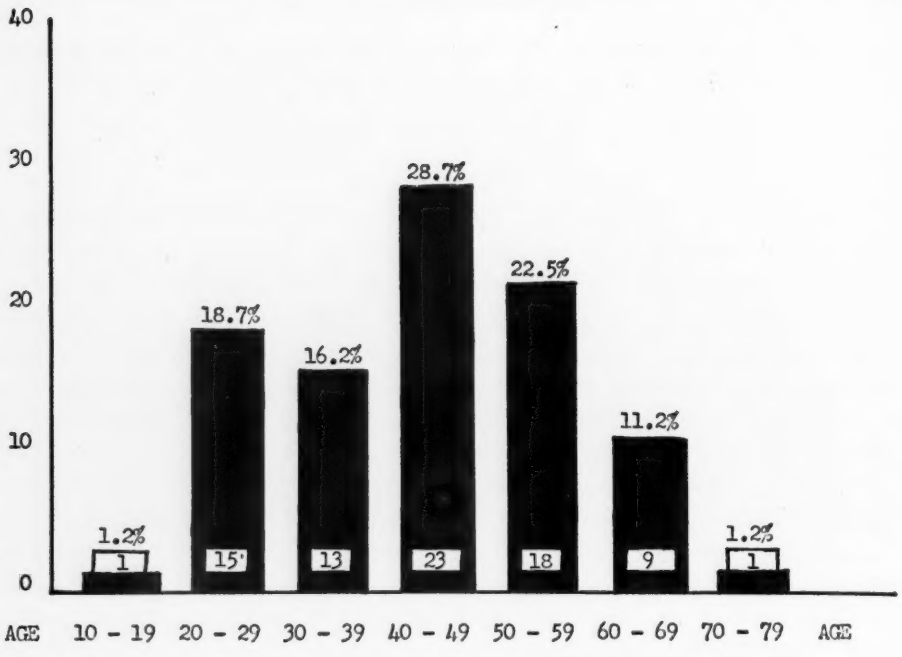

FIG. 1.—Age incidence in decades of acute perforated gastroduodenal ulceration.

POSSIBLE ETIOLOGIC FACTORS	No. of Cases	No. of Cases	Perforation Occurred
1. Relation to food intake	27	22	*16 (72.7%)*. 3 hours or less after eating
			2. 4 hours after eating
			2. 5 hours after eating
			2. 7 hours after eating
		4	Little food intake for 24 hours
		1	While eating
2. At work (physical strain, long hours)	6		
3. During sleep	2		
4. Trauma—struck in abdomen by crowbar 24 hours before perforation	1		
5. Premonitory pain, dull (1, 2, and 5 days duration)	3		
6. Nervous excitement. Spectator at ball game	1		
Total	40		

COMMENT: In 27 the intake of food or the lack of it seemed to play a part. Of 22 patients perforating after eating, in 16 (72.7 per cent) the perforation occurred three hours or less after food intake. In one it occurred while eating, and in four there had been very little if any food intake for 24 hours before perforation. Physical strain while at work or extra long hours of consecutive work apparently was a factor in six. In two the perforation occurred in the early morning hours while asleep. The excitement or nervous tension of a spectator at a close athletic contest was apparently

responsible for one; severe trauma to the abdomen 24 hours before in another. In three there was a constant dull premonitory epigastric pain for one, two, and five days, respectively, before perforation occurred.

While numerous etiologic factors in the production of perforation must be recognized, such as excessive intake of food and drink, extrinsic and intrinsic trauma, infection, excessive use of alcohol and tobacco, and local lack of tissues resistance (Shawan, Fallis, DeBakey, Griswold), more than one may readily be implicated in any given case. In this series, food intake,

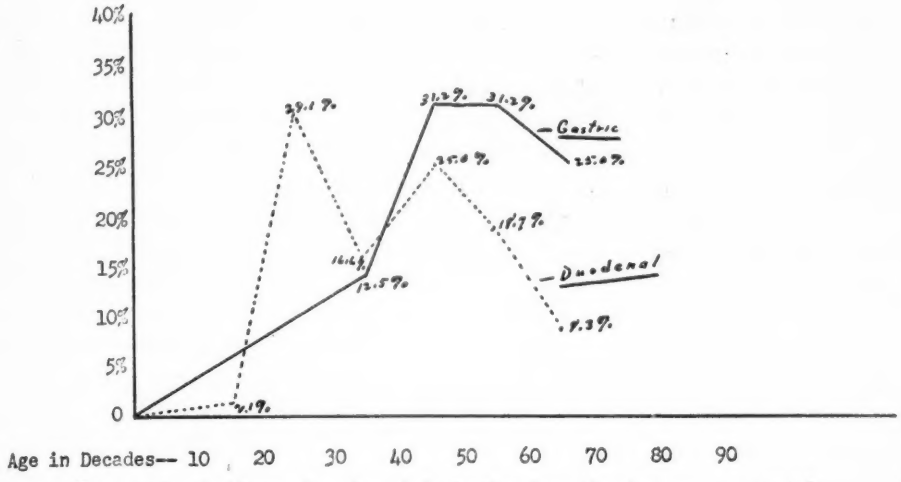

FIG. 2.—Age incidence of perforated duodenal and gastric ulcer cases contrasted.

with its stimulation to gastric motor activity and increase in digestive acids and enzymes, would seem the most prevalent cause of ulcer perforation together with exceptionally long and severe physical strain.

III—PATHOLOGY

Of the 80 cases in this series, there were 63 duodenal, 16 gastric, and one jejunal ulcer, a respective incidence of 78.8, 20, and 1.2 per cent. Of 25 in which cultures from the peritoneal fluid at the time of operation were obtained, eight were positive—two showed *B. coli,* three *nonhemolytic Streptococci,* two *Staphylococci,* and one *Micrococcus catarrhalis.*

IV—SYMPTOMATOLOGY

A. *General.*—Of the 80 patients, all presented a history of abdominal pain, sudden in origin, and of great severity. The symptoms in approximately 90 per cent of the cases were of dramatic onset with marked prostration. Agonizing or excruciating pain was most frequently localized in the epigastrium. As a rule the maximum intensity of the pain was promptly attained, but at times it became progressively more intense, apparently when there was leakage down the right lumbar gutter. The pain was unrelenting, never with a

complete remission, and usually intensified by any movement, deep respiration, or coughing. Cases with free perforation and excessive amount of peritoneal fluid had pain radiating to the shoulder, approximately 50 per cent. 91.2 per cent of the patients in this series presented an abdominal picture of scaphoid abdomen, board-like rigidity, and extreme involuntary muscle spasm. In the remaining 8.8 per cent without rigidity at operation, it was found that the perforation had been plugged by exudate, the omentum, or by an adhesion to an adjacent viscus, and leaking had temporarily been arrested.

B. *Preperforation Bleeding.*—The patients who returned for follow-up examinations were carefully questioned. 26.8 per cent gave a history of evidence of gastro-intestinal bleeding prior to the initial perforation. Several patients who had noticed dizziness, on subsequent questioning recalled having had a tarry stool in association. *Very few* (two) presented evidence of bleeding after the pain of perforation occurred.

C. *Previous Symptoms of Ulcer.*—Of the 80 cases, there were three (3.7 per cent) who gave no history of antecedent peptic ulcer symptoms prior to the onset of pain of perforation. Two of these patients have been questioned in the present follow-up and again deny that they had noticed any symptoms referable to their digestive systems. This incidence of patients without previous ulcer symptomatology is low because exceptionally careful inquiry with regard to previous complaint has been made. Many patients who are writhing in the pains of an acute perforation fail to give accurate histories; and as their previous gastro-intestinal disturbances are so mild in comparison with the type of symptoms which they present with an acute perforation, they fail to recall any preceding discomfort. The remaining 77 cases had durations of symptoms ranging from two weeks to 40 years, with a mean average for the group of 4.8 years.

Study of the relative duration of symptoms in perforated gastric as contrasted with perforated duodenal ulcers reveals a striking difference in the older age group. Perforations in individuals over 50 years of age are more likely to be duodenal if the duration of ulcer symptoms has been over five years and gastric if under five years; a finding which could be suspected, inasmuch, as the onset of duodenal ulcer symptoms is usually in the second or third decade and that of gastric ulcer, not infrequently after 40 years (Fig. 3).

D. *Blood Pressure.*—Of the 77 cases who have records of blood pressure on admission, no systolic values were found to be below 80 Mm. of mercury. In 47 the systolic pressure was between 110 and 130; in 14, over 130 (*i.e.*, in 76 per cent 110, or over); in 16 the systolic pressure was under 110, of which only six were under 100 (7.8 per cent). Profound shock has not been found to occur in any single instance in this series. However, practically all our cases were first examined at least two hours after perforation. One of our patients was seen within 20 minutes following perforation. In this case there was a transitory period of pallor and sweating with mild shock,

but the blood pressure did not fall below 90 systolic, following which the pulse was full, not accelerated, and the systolic pressure rose to 120 and remained at this level until operative intervention. It is possible that if shock occurs following perforation of a peptic ulcer it is transitory and that by the time patients usually reach the hospital it is not in evidence. Our experience indicated that the patients entering the hospital two or more hours after perforation of an ulcer are not in shock, and low systolic blood pressure is rare. Likewise, the pulse is full and the rate is seldom markedly elevated. Fallis also has reported that in his series 55.1 per cent had a pulse rate under 100.

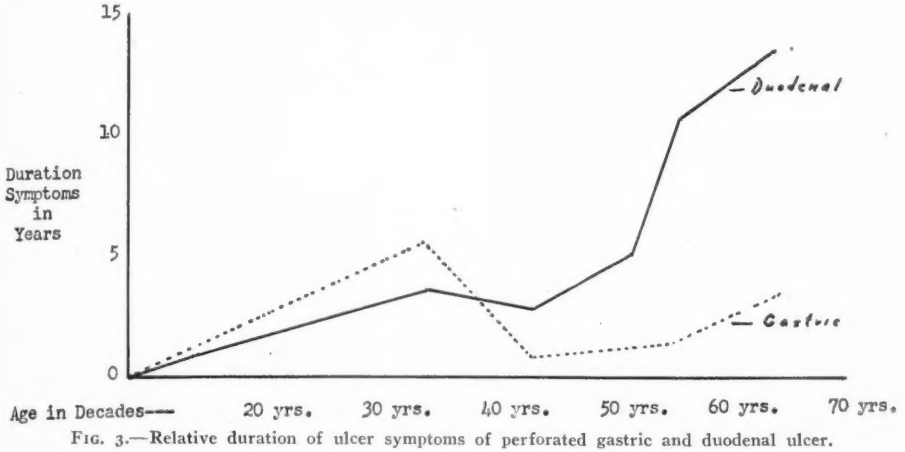

FIG. 3.—Relative duration of ulcer symptoms of perforated gastric and duodenal ulcer.

E. *White Blood Count.*—In 65 of the 80 patients whose leukocyte counts were recorded, the average white blood count was found to be 15,800. The 15,000 to 20,000 group contained the largest number of cases. Only nine, or 11.25 per cent, had counts under 10,000. It is apparent that irritation of the peritoneal tissues by leakage into the peritoneal cavity from a perforated viscus usually provides a decided stimulus to rapid development of leukocytosis. The increased leukocyte count may, therefore, be an early laboratory finding.

V—DIAGNOSIS

The severe agonizing pain, the scaphoid abdomen often with a transverse depression across the epigastrium (Deaver, Estes), board-like abdominal rigidity with extreme tenderness especially in the epigastrium and the full, not accelerated, pulse is a clinical syndrome not simulated by any other acute abdominal accident. However, when the perforation is temporarily plugged by omentum or the leaking is arrested by a plastic adhesion of a nearby viscus the characteristic clinical picture may not be present nor the signs clear.

An aid to diagnosis in the doubtful or atypical case is the demonstration roentgenologically of pneumoperitoneum. As the reliability of the roentgenographic confirmation of the presence of free air under the diaphragm is now well recognized, it should be considered an important diagnostic adjunct.

Of the 47 cases of this series who had roentgenographic or fluoroscopic examinations performed, air was found under the diaphragm in 34 cases, an incidence of 72.3 per cent; and a negative finding was reported in 13 cases, or 27.7 per cent (Fig. 4). There did not seem to be any relationship between the duration of the perforation and the positive finding of pneumoperitoneum.

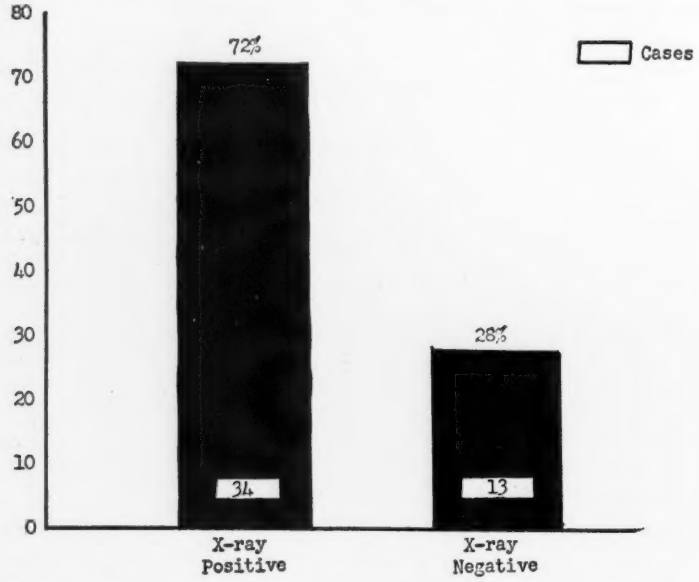

FIG. 4.—Incidence of roentgen ray evidence of pneumoperitoneum in acute perforated gastroduodenal ulceration.

DeBakey, in reviewing the literature, reported a positive incidence of 69.4 per cent in a collected series, and a positive incidence of 64.6 per cent in his own series. In cases in which the roentgenogram does not disclose a pneumoperitoneum, Ochsner and DeBakey have suggested a method for increasing the frequency of obtaining positive films: The stomach contents are first aspirated through a stomach tube, and then a small amount of air, 20 to 30 cc. is injected through the tube. The patient is allowed to rest in the left lateral decubitus position and is fluoroscoped. After a few minutes, the patient is placed in a sitting position and another examination with the fluoroscope is made. If the wall of the stomach or duodenum is perforated, air will be found under the diaphragm. In this series, this test has not been carried out, but the rationale seems sound, especially in those cases where the diagnosis is questionable.

VI—TREATMENT

A. TYPE OF OPERATION

Type of Operation	Cases	Per Cent
Simple closure	61	76.3%
Excision and closure or pyloroplasty	15	18.7%
Closure and gastro-enterostomy	4	5.0%

Simple closure of the perforation was done in 61 cases (76.3 per cent);

excision of the ulcer plus closure or pyloroplasty, 15 cases (18.7 per cent); and simple closure plus gastro-enterostomy in four (5 per cent). For the last ten years simple closure alone has been done. We hold, with Graham, that the patient with a perforation should be treated solely for the lesion creating the emergency. The responsibility of the surgeon is to save life and

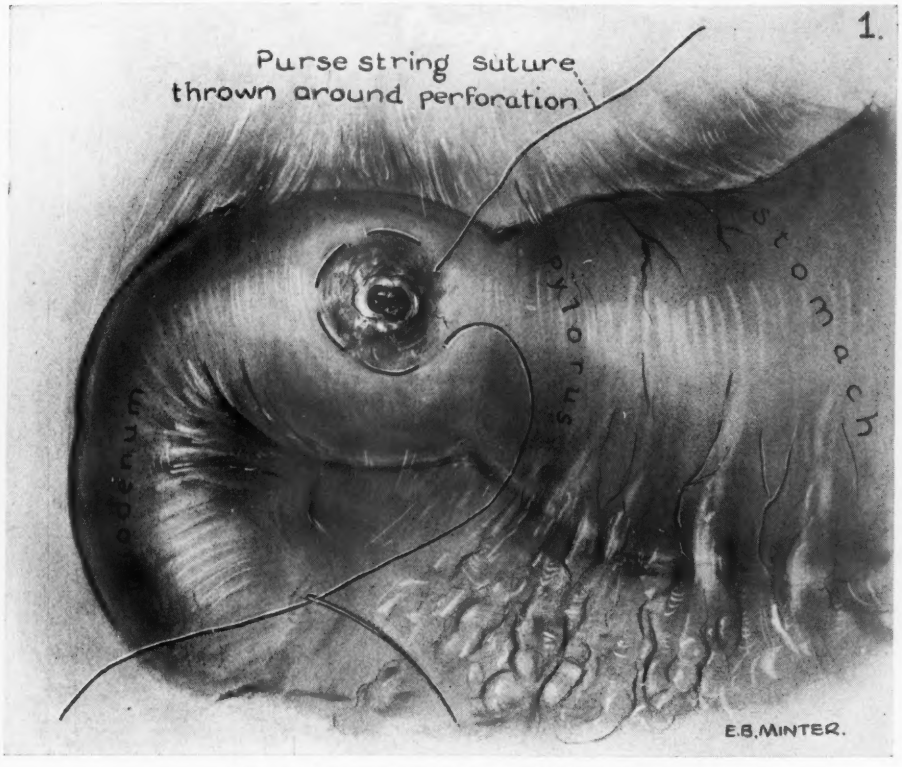

FIG. 5.—Closure of perforation of duodenal ulcer.

the simplest procedure that satisfactorily meets the emergency is the operation of choice. In the face of perforation, the surgeon should be primarily concerned with the patient's recovery from the intra-abdominal catastrophe and not from the ulcer.

B. *Technic of Operation.*—(Simple Closure): The technic we have employed for simple closure of the perforation is very similar to that described by Gatch and Owen. Spinal anesthesia is the anesthetic of choice. An upper right rectus incision gives a quick and adequate exposure. After aspirating any fluid in the subhepatic space, the approach to and ease of manipulation of the ulcer area may be facilitated by the introduction of a stay-suture through the superior border of the duodenum or stomach at the level of the perforation. A purse-string suture of No. 00 chromic catgut is then thrown around the perforation at a distance of at least 1 to 1.5 cm. from it. This is tied snugly, not with great tension, simply to close off the perforation and prevent further leakage (Fig. 5). Then the ulcer area is buried, enfolded,

and pushed backward beneath two layers of No. 00 chromic catgut sutures, placed transversely to the long axis of the intestine beginning at the margin of the gastrohepatic omentum, and in the case of a duodenal ulcer suturing stomach wall to duodenal wall (Fig. 6). There is little tendency to narrow or stenose the lumen of the duodenum by these sutures; the ulcer area is merely enfolded and pushed backward and the perforation well closed and

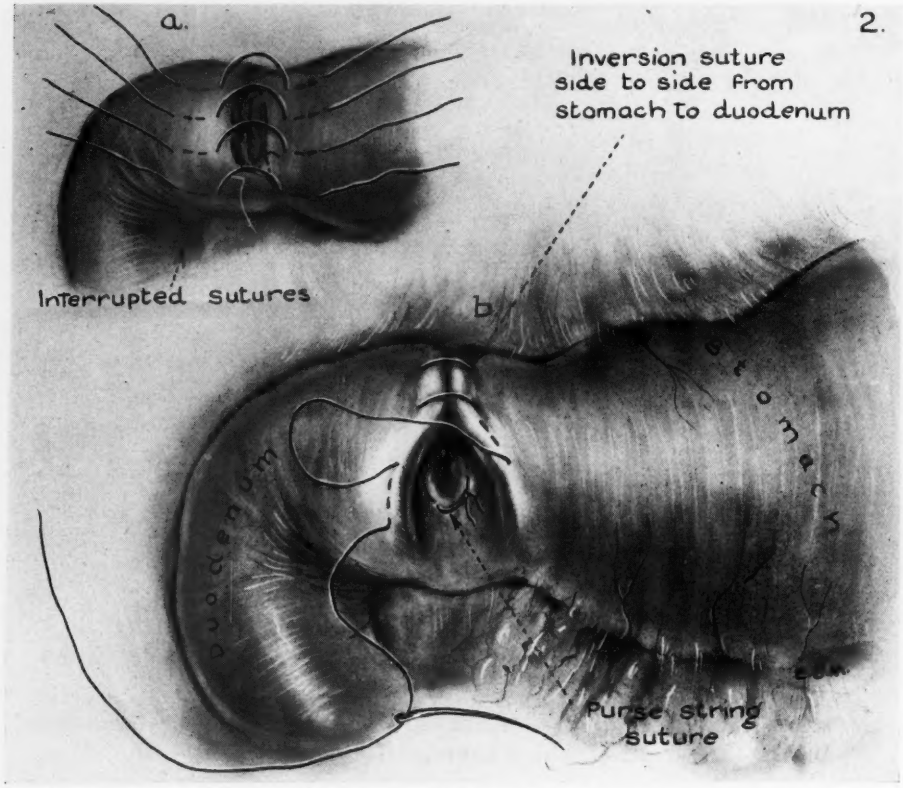

FIG. 6.—Closure of perforation of duodenal ulcer.

covered (Fig. 7). Sulfanilamide may be dusted over the suture line and in the subhepatic space. Finally, whenever possible, the tip of the greater omentum is placed over the operative site. Drainage is only used in the late cases and when, because of the size of the ulcer, or perforation, or extensive induration, completely satisfactory enfolding by suture has not been obtained and further leakage might be possible. Drainage in this series was not used in 34 of 38 cases where operation was performed within six hours of perforation.

VII—MORTALITY

In the total series of 80 cases there were seven postoperative deaths, a mortality rate of 6.7 per cent. Following simple closure in 61 cases (76.2 per cent), there were five deaths, a mortality rate of 8.2 per cent. Excision

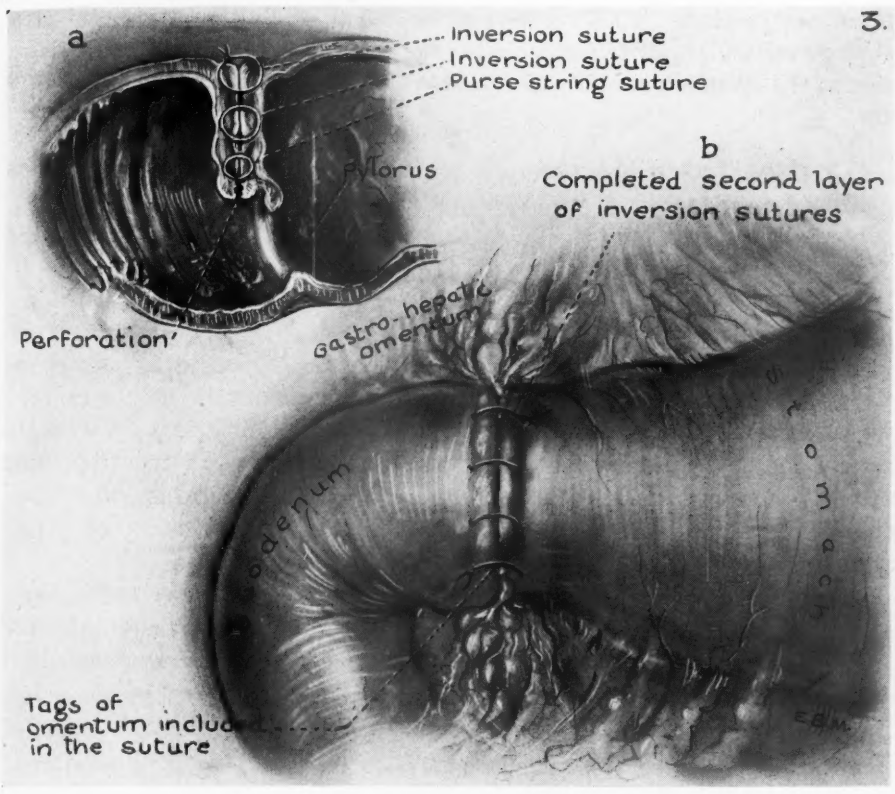

FIG. 7.—Closure of perforation of duodenal ulcer.

A. TYPE OF OPERATION

Type of Operation	Cases	Deaths	Mortality Rate
Simple closure	61	5	8.2%
Excision and closure or pyloroplasty	15	2	13.3%
Simple closure and gastro-enterostomy	4	0	0.0%

plus closure by pyloroplasty was employed in 15 cases (18.7 per cent), with two deaths, a mortality rate of 13.3 per cent. Simple closure plus gastro-enterostomy was utilized in only four cases (5 per cent), with no deaths. The lower mortality rate in the simple closure plus gastro-enterostomy may be due to the fact that these patients were considered to be in so satisfactory condition that more extensive measures seemed justified.

Similarly, in a collected series of 11,284 cases reported by DeBakey, the mortality following simple closure was 25.9 per cent; following closure plus gastro-enterostomy, 20.4 per cent; and following partial gastrectomy, 13.4 per cent. Unquestionably, the better risk patients were subjected to the more extensive procedures which accounts for the lower postoperative mortality of gastro-enterostomy and gastric resection. This, however, should not be a valid argument for their more frequent use in the presence of a perforation. Even in the face of apparent lower mortality and even though partial gastrectomy affords a greater chance for freedom from subsequent gastro-

intestinal symptoms, it should be reserved as an operation of election at a later period when full recovery from the acute emergency has taken place and not the operation of choice at the time of the perforation.

B. DURATION OF PERFORATION

Duration of Perforation in Hours	Cases	Deaths	Mortality Rate
0- 6 hours	38	0	0.0%
6-12 hours	17	2	11.7%
12-18 hours	10	0	0.0%
18-36 hours	6	2	33.3%
36- hours	9	3	33.3%
Total	80	7	8.7%

There was no mortality in the 38 cases operated upon within six hours of perforation. Inasmuch, as this comprised approximately 50 per cent of the cases, it is readily recognized that the low mortality in this series is largely due to early operative interference. 33.3 per cent of those operated upon 18 hours or more after perforation died, indicating, as most other observers have noted, that the mortality following perforation of peptic ulcer lies largely in delay in operation.

The primary causes of death in fatal cases are acute peritonitis, subhepatic abscess, or pulmonary complications. In the seven deaths, three autopsies were obtained. In one, the lethal lesion was diffuse peritonitis; in the second, subhepatic abscess and basal pneumonia; and in the third, diffuse peritonitis, with consolidation in both lower lung fields.

C. TYPES OF ANESTHESIA

Type of Anesthesia	Cases	Deaths	Mortality Rate
Spinal	70	5	7.5%
General	9	1	11.1%
Local	1	1	100.0%

Of the 80 cases, 70 received some form of spinal anesthesia; nine received general anesthesia; and one, local infiltration anesthesia, with mortality rates of 7.5, 11.1, and 100 per cent, respectively. Fallis reports a mortality incidence of 7.3 per cent for spinal, 29.3 per cent for general, and 100 per cent for local anesthesia. Shawan's figures indicate that the mortality rate following general anesthesia was twice that of the spinal group. Obviously, local anesthesia is used only in the very bad risk cases and high mortality is to be anticipated.

Although the number of our cases who had general anesthesia may be too small upon which to base accurate conclusions, a comparison of the post-anesthetic complications of spinal and general anesthesia would seem of interest. There were no deaths at the operating table or immediately post-operative with any of the anesthetics employed. In the nine cases with general anesthesia, there were three respiratory complications—two pneumonias and one empyema, an incidence of 33.3 per cent. In the spinal group of 70 cases, there were four pneumonias, one bronchitis, and one empyema, or a total of six complications above the diaphragm, an incidence of 8.5

per cent. The intraperitoneal complications following general anesthesia were two subphrenic abscesses, one subhepatic abscess, or three out of nine cases, an incidence of 33.3 per cent. After spinal anesthesia, two drainage tract abscesses, three subphrenic abscesses, and five cases of diffuse peritonitis, an incidence of 14.2 per cent.

Graham believes that spinal anesthesia is preferable because the pronounced respiratory efforts produced by general anesthesia causes a plunger action of the diaphragm on the liver which tends to disseminate intraperitoneal exudate and leakage over a wider area.

COMPARATIVE MORTALITY RATE

Name	Cases	Mortality Rate
Shawan	387	25.43%
DeBakey (collected series)	15340	23.4 %
(own series)		18.2 %
Eliason and Thigpen	78	21.43%
Guthrie and Sharer	78	20.8 %
Fallis	100	20.0 %
Shelley	82	18.2 %
Griswold	111	18.0 %
Paletta and Hill	83	16.9 %
Estes and Bennett	80	8.7 %
Graham, R. R.	62	3.2 %

The relatively low mortality rate in our series would seem to be due largely to the high percentage operated upon early, 50 per cent within six hours of perforation; 81 per cent within 18 hours. Spinal anesthesia may also have played a part, as 87.5 per cent had a spinal anesthetic. It has been our routine procedure to begin intravenous therapy, either plasma or 5 per cent glucose, on the operating table immediately following the administration of the anesthetic. Graham feels that, particularly in the late case, appropriate intravenous therapy preoperatively has been a factor in the low mortality of his series. Likewise, postoperative gastric suction and sulfonamide therapy should aid in mortality reduction (Ulfelder and Allen, Griswold). The mortality following gastric perforation in our series was 12.8 per cent, two deaths in 16 cases, and 7.9 per cent following duodenal perforation, five deaths in 63 cases. This greater mortality following gastric perforation is in agreement with most previous statistics. However, our gastric cases were relatively few.

VIII—END-RESULTS

Of the 80 cases of gastroduodenal ulceration in this series, there were seven postoperative deaths, with 73 cases remaining. Of this number, no follow-up reports were available on ten, and there were ten known subsequent deaths. This leaves 53 cases whose course has been satisfactorily determined for statistical study.

There were 61 cases of simple closure, with five operative deaths. Of the remaining 56 cases, it has been impossible to trace six. Of the 50 patients whose subsequent course has been determined, 11 (22.0 per cent) have had subsequent operations for gastric or duodenal pathology, seven

A. TYPE OF OPERATION

Operation	Cases	Followed	Reoperation	
Simple closure	50	11–(22.0%)	4—gastro-enterostomy or resection	8%
			7—reperforation	14%
Excision and pyloroplasty	11	2–(18.1%)	1—reperforation	9%
			1—gastro-enterostomy	9%
Closure and gastro-enterostomy	2	0– (0.0%)	1—case rehospitalized for control of symptoms	

cases being for reperforation. Of the 15 cases treated at the time of initial perforation by the operation of excision plus pyloroplasty, there were two postoperative deaths. No follow-up records are available on two. Thus, 11 cases have been followed. Of these, one has reperforated, and one has had a gastro-enterostomy performed. Therefore, of the 11, two have been subjected to reoperation (18.1 per cent). Of the four patients having simple closure plus gastro-enterostomy, the records of two are not available due to loss of contact with the patient. Of the remaining two, neither has had subsequent operations, although one has had two periods of hospitalization for control of peptic ulcer symptoms.

It would seem, therefore, that recurrence of gastric or duodenal ulcer symptoms may occur after any one of the operative procedures employed for treatment of perforation, and with about equal relative incidence following simple closure or excision and pyloroplasty.

It is to be noted that in the 50 cases of simple closure, excluding the seven cases of reperforation, there were but four cases (8 per cent) requiring subsequent secondary operation as compared to one of 20 (5 per cent) in Gatch and Owen's report; ten of 55 (18.1 per cent) in Guthrie and Sharer's series; 11 of 51 (21.5 per cent) reported by Graham; and 15 per cent as stated by White and Patterson.

B. POSTOPERATIVE SYMPTOMS

The 53 cases with follow-up records have been divided into three groups according to their postoperative clinical course. They have been very thoroughly interrogated and their complaints and symptoms very strictly interpreted. The first group are those who have been apparently "cured" by the original operative procedure; the second group includes those in a satisfactory condition with conservative measures effectively controlling symptoms; and the third group includes those whose course has been entirely unsatisfactory.

FOLLOW-UP RESULTS—53 CASES

Group I:		
"Cured"	3	(5.6%)
Group II:		
Symptoms controlled by medical treatment	38	(71.7%)
Group III:		
Symptoms not controlled by medical treatment	12	(22.7%)
Total	53	

GROUP I.—In this group are included those cases who have presented

no complaints relative to the stomach or duodenum *at any time* since the original operation for perforated ulcer. They have not remained on any dietary restrictions, and have received no alkali, antispasmodic, or other similar medication. This group comprises but three cases. The average time followed was 6.5 years and the average age was 54 years. The site of perforation was duodenal in all three cases. The types of operation performed were simple closure in two instances and simple closure plus gastro-enterostomy in the third. Two perform heavy work, one a farmer and the other an industrial laborer. The third is unemployed. All three state that they do not smoke; two deny indulgence in alcohol, while the third states that he has no untoward gastro-intestinal symptoms following the use of alcohol.

In comparison with the end-results of others (Shelley, and White and Patterson report that as high as 44 and 60 per cent, respectively, remain well and free of symptoms), 5.6 per cent cured of this series is an exceedingly low figure; in all likelihood the result of strict interpretation and the inclusion of even minor complaints as indicating possible residual disease.

GROUP II.—Included in Group II are all patients who, at any time subsequent to perforation, have found it necessary to utilize any one, or any combination of, the following measures for the control of symptoms: (1) Alkali; (2) restricted diet; (3) antispasmodic; and (4) sedation. Of the 53 cases followed 38 were placed in this group. The site of lesion was duodenal in 33 cases and gastric in five cases. Thirty had simple closure, six had excision plus closure or pyloroplasty; and two had simple closure plus gastro-enterostomy. Of the 29 who smoked, 12 stated that their gastroduodenal symptoms were increased following excessive use of tobacco. Nineteen of the 38 gave history of unexplained periods of exacerbations of symptoms, with spontaneous remission. There was history of postoperative melena in three of the 38 cases. The type of work was heavy in 21 cases, sedentary in eight, moderately active in seven, and two were unemployed.

Twenty of the 38 had voluntarily sought a physician's care for gastroduodenal symptoms at some time subsequent to the operation for perforation. The earning power of 16 of the 38 cases had been impaired due to the patient's gastro-intestinal complaints.

Eleven were noted to have the onset of symptoms while still on convalescent ulcer diet and eight noted onset of symptoms immediately after a resumption of full diet. Three of the 38 cases have followed a careful postoperative medical regimen and have never complained of gastro-intestinal symptoms, but they have not attempted to eat all types of food.

End-results of this group may be summarized as:

(a) Patients who are easily controlled by one or a combination of alkalies, diet, or medication and those who only occasionally require dietary or medicinal measures for relief—17 cases (44.7 per cent).

(b) Those who *quite frequently* require diet and medication for relief of periodic complaint—18 cases (47.3 per cent).

(c) Those patients whose symptoms are controlled with considerable difficulty. These usually employ all the dietary and medicinal measures in order to control their gastro-intestinal symptoms—three cases (8 per cent). It is to be noted that four of this group, while apparently well controlled, reperforated.

GROUP III.—Twelve cases are classified in Group III. These comprise those who had gastroduodenal symptoms to a marked degree which were not kept in abeyance by any of the conservative measures, and also patients who are at present *symptom-free* but who required *operative measures for relief* of their *persistent symptoms subsequent* to the *first perforation.* Nine of the 12 patients stated that their symptoms had been intensified by excessive smoking. Five confessed to an overindulgence in alcohol, with an accompanying increase in digestive complaints. All 12 described their most prominent symptom as upper abdominal pain with the stomach empty. Eight of the 12 gave a history of spontaneous remissions with subsequent acute exacerbations. All 12 stated that certain types of food, usually heavy, greasy, or highly seasoned, would produce symptoms. Of the 12, eight had been engaged in sedentary occupations and one, moderately heavy work. In ten of the 12, the earning power had been impaired by the digestive symptoms, and eight of the 12 had engaged in irregular hours of work, changing shifts, etc. Eleven of the 12 patients remained on a convalescent ulcer diet following discharge from the hospital. Of these, three patients had onset of symptoms while still carefully following this regimen. Four began to have symptoms with resumption of a more liberal full diet.

Four of the 12 reperforated. Of these four, in one, after an asymptomatic period of two postoperative years, the first symptom was that of the pain of reperforation.

Four of the 12 have had subsequent elective surgery; two had a gastroenterostomy for symptoms of long-standing pyloric obstruction; and two have had partial gastric resection for persistent pain from an active duodenal ulcer. All of these are at present relatively asymptomatic on conservative dietary management without antispasmodic or alkali adjunct therapy.

Four of the 12 were rehospitalized and had their symptoms controlled by energetic medical treatment, which all have followed to date, and all report themselves fairly well controlled. One patient has persistent epigastric pain which has been partly incapacitating even though he has carefully maintained a strict dietary and medical management at home. He has refused hospitalization.

SUMMARY OF END-RESULTS IN GROUP III:

Reperforation	4
Subsequent elective surgery	4
Rehospitalized for medical control	3
Treatment refused	1
Total	12

IX—REPERFORATION

Of the 73 cases who were discharged from the hospital we have been unable to follow ten. Sixty-three have been followed sufficiently to obtain a report on the late results. Of these, eight developed reperforation; six were duodenal and two gastric. Seven occurred in cases that had had simple closure of the original ulcer and one following excision and pyloroplasty.

The site of the ulcer of the second perforation *was the same as in the initial perforation* in three, two of these cases being gastric and one duodenal. The remaining five very definitely had an ulceration at a different site from that of the first perforation. The onset of ulcer symptoms in four cases occurred approximately two years following the initial perforation when apparently the patient had been satisfactorily controlled by diet and alkali and had been classified in Group II. These symptoms were the pains of the second perforation. Two began to have recurrent symptoms immediately following their first perforation and persisted for one month and five years, respectively, before the second perforation occurred. Apparently reperforation may, therefore, develop both with a slow insidious onset or with a rather sudden flare-up without premonitory symptoms. One patient with a duodenal ulcer reperforated twice after simple closure in each instance.

Of the eight cases, three presented definite histories of bleeding prior to the onset of the second perforation. One patient noted bloody vomitus six months before his second perforation occurred; the other two, tarry stools within a short period of time prior to reperforation.

Incidence of reperforation in this series (12.7 per cent) is exceedingly high. Of 4,183 cases of perforated ulcer reviewed by Pearse, only 33 reperforated, or 0.69 per cent; of DeBakey's 6,538 collected cases, 74, or 1.1 per cent; and nine of Ulfelder and Allen's 334 cases (2.7 per cent). Pearse believes these figures are low. He found that reperforation occurs after all of the operative procedures used for treatment and about equally in the relative proportion of their use. Repeated reperforation is less common after simple closure than after closure plus gastro-enterostomy.

Inasmuch, as the cause of reperforation in peptic ulcer has been largely conjectural, it seemed of interest to review the records of these reperforated cases to ascertain if any possible etiologic factor might be recognized:

(a) *Occupation.*—The majority of the patients in this group were found to do heavy types of physical work. Five worked on eight-hour shifts which varied each week and consequently could not form regular dietary habits.

(b) *Alcohol and Tobacco.*—Of the eight patients, four were known to indulge to excess in alcoholic beverages at frequent intervals, with a resulting exacerbation of their ulcer symptoms. Five apparently smoked excessively, and three stated that their symptoms were increased by overindulgence in cigarette smoking and were attenuated after reduction in the amount of tobacco used.

(c) *Subsequent Symptoms.*—The five patients still living are employed

at present but are not undertaking any exhausting physical types of work. All are taking frequent feedings throughout the waking hours. Three find that milk helps to control symptoms. Four are satisfactorily controlled by a careful dietary regimen plus alkali plus antispasmodics. The remaining patient has periodic attacks of epigastric pain with remissions. He has refused roentgenologic study to date.

(d) *Gastric Analyses.*—Postoperative gastric analyses, obtained in six of the eight cases, all presented readings far above the normal range. The maximum free hydrochloric acid values were 50, 135, 86, 78, 94, and 98, with an average value of 90 for the group. The test meal with histamine was the method utilized in each case except one, where the alcohol test meal was employed. Four analyses were taken following the second perforation which indicated hyperacidity values approximating those following the first.

A comparison of the average acids in the reperforation group with those with but a single perforation is illuminating. The average maximum free hydrochloric acid value for 21 of the single perforation cases was found to be 70. The patients who *reperforated,* therefore, showed higher acid values than those who did not.

It would seem that these exceedingly high acid values of the reperforation group are not merely incidental findings but represent a possible element of definite pathology which may be in part responsible for the development of subsequent ulcerations and possibly repeated perforation.

(e) *Mortality.*—Three patients have died subsequent to discharge from the hospital; a 55-year-old man was one, who succumbed to an acute gastrointestinal hemorrhage six months following his reperforation. He had noted recurrent epigastric pain soon after leaving the hospital and had not sought medical aid for relief of symptoms. The second was a 61-year-old male who developed evidence of pyloric obstruction and had a posterior gastroenterostomy performed six months after his second perforation, following which he was symptom-free on a bland diet, and died eight years later of coronary occiusion. The third was a man of 29 years of age at the time of his first perforation in 1928. Simple closure was done. He was symptom-free on an ulcer diet until 11 months later, at which time he began to notice return of epigastric pain which was followed in one week by a reperforation of his duodenal ulcer. Simple closure again was employed, the ulcer being in the exact location of the previous perforation. From 1929 to 1940 his symptoms were controlled by a bland diet with frequent feedings. In 1940 he discontinued treatment and in three months developed vomiting and pain. He was treated elsewhere, and a gastro-enterostomy for a duodenal ulcer was performed. In 1941 his symptoms recurred, and he returned to the same hospital for a perforated jejunal ulcer which was treated by plication. His symptoms persisted and two months later he was subjected to a gastric resection from which he failed to recover.

In summary, it may be stated that:

(a) Six of the patients with reperforation were in the 30- to 40-year age group and engaged in heavy physical work.

(b) A majority used alcohol and tobacco to excess.

(c) Six had unusually high free hydrochloric acid values in the gastric analysis.

(d) One patient after reperforating developed a third duodenal ulcer that failed to respond to a gastro-enterostomy and required a gastric resection. One reperforated a second time. One has another active ulcer. One died of a gastro-intestinal hemorrhage. One required a secondary gastro-enterostomy.

It would seem that patients who have had a perforation of a peptic ulcer and (1) have irregular working hours, (2) admit dietary indiscretions, and (3) are shown to have high acid values in postoperative gastric analyses, are all likely to be candidates for subsequent reperforation. It is suggested, therefore, that a routine postoperative gastric analyses should be made in all cases recovering from gastroduodenal perforations. In those cases in which repeated analyses show persistent excessive hyperacidity and particularly those difficult to control by diet, alkalis, or other medication, recurrent ulceration with subsequent reperforation is exceedingly likely; and subtotal gastrectomy would seem definitely indicated, not only to protect the patient against further gastroduodenal ulceration but to avoid the danger of a subsequent reperforation.

SUMMARY AND CONCLUSIONS

1. Eighty cases of acute perforated gastroduodenal ulceration treated by operation are reported, with an operative mortality of 8.7 per cent. Of these, 63 were duodenal, 16 gastric, and one jejunal.

2. The clinical syndrome of perforation, as observed at the time of admission to the hospital, is described. Shock is rarely present; systolic blood pressure in 76 per cent was 110, or over.

3. Diagnosis in the atypical case may be confirmed by roentgenologic demonstration of pneumoperitoneum.

4. Simple closure of the perforation is the operation of choice. The method of closure employed in this series is detailed.

5. Low postoperative mortality rate (8.7 per cent) may be attributed to (a) early operative interference, 81 per cent operated upon within 18 hours of perforation; (b) spinal anesthesia; (c) immediate (preoperative) routine use of intravenous therapy (plasma, glucose, saline); and (d) postoperative gastric suction and sulfonamides.

6. End-results have been reported in detail. Of those followed, only 5.6 per cent were completely cured; 71.7 per cent required medical treatment for control; and 22.8 per cent required further surgery or hospitalization for relief. Perforation, therefore, but rarely confers immunity from further ulcer symptoms. Following operation, perforation cases require the same continuous medical supervision that any peptic ulcer case requires.

7. Four of the 50 cases of simple closure followed required secondary gastro-enterostomy or resection.

8. Recurrent ulceration was observed after all types of operative measures used for the closure of the perforation.

9. Reperforation occurred in eight cases (12.7 per cent)—six duodenal, and two gastric. The common factors that these presented were: (a) Dietary indiscretions. (b) Overindulgence in alcohol and tobacco. (c) Marked gastric hyperacidity.

10. It is suggested that of postoperative perforated ulcer cases, those in which repeated gastric analyses show persistent excessive hyperacidity, and are difficult to control by medical measures, recurrent ulceration with subsequent reperforation is likely and partial gastrectomy would seem indicated.

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DISCUSSION.—DR. FRANK K. BOLAND, Atlanta, Ga.: I wish to say a few words about this disease in the colored race. Some 30 or 40 years ago it was rare in the Negro, but the incidence of the disease, it seems to me, has increased since that time until now it nearly approaches that of the white race. For a long time I have tried to show that this increase is somewhat a matter of diet. When the Negro adhered to his old diet of "pot likker" and similar common foods with a high vitamin C content, there was very little peptic ulcer, gallbladder disease or appendicitis, but since he has become more civilized and eats more white folks' food he has more white folks' diseases.

We have more perforating than nonperforating peptic ulcers at our hospital. This year we have had something like 30 perforations, with a mortality of 20 per cent. Of course many of these cases come in late, but I note Doctor Estes' statistics show about the same ratio. I believe with spinal anesthesia our mortality would be less.

I wish to report one case, that of a Negro who had been sick for four days before entering the hospital. He had had several episodes of epigastric pain during the past few years. When he came to the hospital he was in shock, so much so that while the diagnosis of perforated ulcer was made we could not get him into condition to operate; we gave several blood transfusions and other supporting measures. He had bulging in both flanks and evidently an accumulation of fluid. (slides) This is the roentgenogram, and I would like to call attention to two points; one is the pneumoperitoneum all over the left side; this is very marked and looks like a pneumothorax in the chest. He had more free pus than I have ever seen in the abdomen; about 1,000 cc. was evacuated from each side when the tubes were put in. (slide)

The autopsy showed the largest perforated ulcer I have seen, 2 cm. in diameter. I notice Doctor Estes showed one large perforation in a drawing, but mine is from an actual autopsy specimen and diagnosed by the pathologist as prepyloric ulcer. So this case had this large perforation, a marked amount of pneumoperitoneum and a half-gallon of pus which was aspirated from the peritoneal cavity.

DR. J. M. T. FINNEY, Baltimore, Md.: I think we are all very much indebted to Doctor Estes for a most comprehensive and detailed study. I want to call attention once more, as I did a few years ago, to a very simple method of closure of a ruptured ulcer which was first described, I believe, by one of our Senior members, Doctor Miller. I have employed it in 29 cases in the last eight years.

I think most of us have realized the difficulty of placing sutures in the tissue around the ulcer and have them hold; because of edema and scarring they almost invariably tend to cut out. Also I believe we have been impressed by the danger of constricting the lumen. Doctor Estes felt he could close without too much constriction—I have not had that luck.

The method advocated consists in dividing and detaching the round ligament of the liver from the under surface of the abdominal wall near the umbilicus; it is a rather fibrous structure in most people. If one then takes the cut end of the round ligament, leaving the upper end attached to the liver, and sutures it tight against or even into the perforated ulcer, and for about one centimeter around the perforation, there is a solid plug which will close the hole and effectually cover the wall around the perforation. One does not have to pull the sutures to close the hole to make it secure, so the sutures do not tend to cut out; it is easy to place a plug to cover a considerable area and in that way to block off not merely the perforation but a large area around it.

Using that method, in 29 cases I have had one death in the last eight years, and that was a perforated gastric carcinoma. I have employed it in one gastric ulcer and 27 duodenal ulcers. To date I have not had occasion to reopen any of these cases for reperforation or for stenosis. Whether they have gone elsewhere for a second operation I do not know, because I have not followed them all. Most of them came from around Baltimore and I believe I should have heard had any come to second operation. I realize one is skating on thin ice and no doubt there will be secondary operations, but personally I have had more satisfaction with this procedure than with any other.

DR. CLAUDE J. HUNT, Kansas City, Mo.: I have enjoyed this presentation by Doctor Estes. He has covered the subject so thoroughly that there is little to be said in discussing his paper. He has emphasized the relative frequency of pneumoperitoneum in the perforation of peptic ulcer. We think this is a positive diagnostic procedure and

is of great value when it is present. If the perforation is free into the abdominal cavity one can usually find evidence of pneumoperitoneum. If the patient is elevated and in somewhat a sitting position, air can usually be seen between the liver and the diaphragm. However, the absence of free air in the abdominal cavity should not cause one to delay operation if there are clinical manifestations of perforation.

We feel that at operation only such measures should be taken as will result in the restoration of the patient to health and save his life. The situation is a tragic one, accompanied by shock, and demands immediate operation upon a patient unprepared for major surgery. Therefore, we believe that simple closure and nothing else should be attempted. We believe that further surgery should be deferred until the patient is sufficiently recovered from the tragedy and the shock so that he may be adequately prepared for a gastric resection. It is rarely necessary to do a gastro-enterostomy at the time of preparation. We have felt it necessary to do so only twice in our experience. We have not done a partial gastrectomy at the time of primary closure. We have, however, on several occasions, reopened the abdomen at a later date and done a partial gastrectomy.

The question of frequent perforations at the same site is emphasized in the following case: A boy, at the age of 17, had the first perforation, and at the age of 23 had the fourth perforation. The fourth one we closed; the three previous ones were closed elsewhere. Subsequent to the closure of the fourth perforation we did a partial gastrectomy from which he recovered nicely and has remained well to date. This emphasizes the fact that one may have multiple perforations and that with more than one perforation gastric resection should be carried out.

DR. R. M. POOL, Fairfield, Ala.: At the Employees' Hospital at Fairfield, where Dr. Lloyd Noland is my chief, we have observed 50 perforated peptic ulcers during the past eight years. In this group of 50 cases, two had reperforations. At the operation for second perforation a simple closure was done and we have not heard from these since. An interesting thing is the interval between the first and second perforations; one occurred in about a year at the site of the previous ulcer, and the other after about 18 months.

Three cases of perforated peptic ulcer came to secondary operation. In this group of three, a simple posterior gastro-enterostomy was done. We realize that gastro-enterostomy has certain limitations but we felt in these cases that it was the operation of choice because of pyloric obstruction with gastric retention and because of involvement of the head of the pancreas.

In our series of 50 cases there were seven deaths. We thought this was rather high, but after seeing Doctor Estes' figures we find it is not so high. Out of the seven deaths we would like to eliminate three from the statistics. One was a white woman, age 78, who was admitted three days after perforation in very poor condition, and who died without operation. The clinical diagnosis in this case was perforation of the stomach due to malignancy, but postmortem examination showed a perforated, benign duodenal ulcer, with peritonitis. Another patient died on the operating table and we considered that an anesthetic death. The third, who also died on the operating table, was practically moribund at the time of admission and should probably not have been subjected to surgery.

DR. W. L. ESTES, JR., Bethlehem, Pa. (closing): The large perforations are more likely to be attended by shock, more likely to be followed by rapid fulminating peritonitis, and require especially prompt operative interference. In these cases the problem of adequate closure may be more difficult to meet. The method of closure of the perforation is always of interest. Doctor Finney spoke of his very ingenious method and the end-results. I think most of us are familiar with the method suggested by Roscoe Graham, of simply taking a free bit of omental graft and suturing it over the opening; this simple procedure was employed in the series in which his very low postoperative mortality was obtained. Whatever technic is used, it is important that the closure be tight and complete and that an adequate lumen of the duodenum be preserved. In the type of closure we described I want to emphasize two things: The purse-string suture should not be tied too tight; the infolding sutures can be taken at quite a distance from the perforation. Even with large infolding there is little danger of stenosis.

There should never be any delay in operation in order to obtain a radiograph to prove the presence of pneumoperitoneum; with a clear-cut clinical syndrome immediate operation is demanded with no time out for unnecessary diagnostic refinement.

Reperforation occurred in our series as early as six weeks following the first perforation, and as late as four years. Three cases reperforated two years after the original perforation with no premonitory symptoms of any sort. Reperforation unquestionably is of more frequent occurrence than it has been hitherto statistically indicated. Perforation does not necessarily confer immunity from further ulcer development nor alter the "ulcer diathesis." Therefore, ulcer cases after perforation should have the same careful persistent medical supervision that any peptic ulcer case demands.

CARCINOMA OF THE DUODENUM*

REPORT OF TWO CASES IN SUPRAPAPILLARY PORTION

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NEW ORLEANS, LA.

THIS REPORT is based on two cases of carcinoma of the suprapapillary portion of the duodenum. A correct preoperative diagnosis was made in both instances. It should be stated, however, that the diagnosis was largely based upon the roentgenologic findings.

Case 1.—C. M., white, female, age 71, married, was admitted to Charity Hospital of New Orleans, January 31, 1942, complaining of "a lump in the stomach."

Two years prior to admission she began to vomit, and stated that the vomiting was associated with headaches. About one month prior to admission, she noted a mass in the epigastrium just to the right of the midline. She would vomit from time to time, but the vomiting was not associated with nausea. She complained of pain in the upper right quadrant of the abdomen. There had been a weight loss of from 20 to 30 pounds during the previous two months. The bowel habits had been regular and at no time had she tarry stools.

In briefly summarizing the findings only the essential facts in connection with the examination which have to do with the abdominal findings will be given.

The abdomen was soft, slightly rounded. The veins were not prominent. There was tenderness in the epigastrium and to the right of it. To the right of the midline in the epigastrium a movable, tender, hard, irregular mass was palpable. The liver and spleen were not palpable. Stools were negative for blood, parasites, and pus. Gastric analysis: Occult blood, no free hydrochloric acid, total acidity of 6.

Roentgenologic Examination.—G. I. tract, No. 655332. February 3, 1942: "There is a constant defect in the distal portion of the stomach and proximal portion of the duodenum. At the end of six hours, there is 40 per cent gastric retention. The findings are compatible with carcinoma of the stomach or pancreas."

A review of the roentgenograms which suggested the diagnosis of carcinoma of the duodenum revealed the following: 1. The first portion of the duodenum was dilated, and even after the barium had passed into the small intestines, there was evidence of some retention of the barium on a changed mucous pattern of the duodenum. It seemed that this could only be due to a fungus-like growth or possibly a polypus within the duodenum around which the barium remained adherent. We felt inclined to the diagnosis of carcinoma of the duodenum and the operative findings proved the correctness of the assumption.

Operation.—February 11, 1942: *Preoperative Diagnosis:* Carcinoma of the duodenum in the suprapapillary portion.

Summary of Operation.—A right paramedian incision was made from the costal margin to just below the level of the umbilicus, no free peritoneal fluid was encountered.

A firm mass, about 5.5 cm. in diameter, was found in the first portion of the duodenum, just distal to the pylorus. The liver was inspected and palpated, and no evidence of metastases were found. Some nodules were found in the great omentum close to the greater curvature of the stomach. The head of the pancreas was intimately adherent to the involved portion of the duodenum, and was apparently involved in the disease. We

* Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

FIG. 1.—Roentgenogram showing mucosal pattern changes in the duodenum.

were able to mobilize the duodenum and section it below the growth. The distal stump of the duodenum was closed with tier sutures, after the Parker-Kerr technic. Resection of the stomach and duodenum was proceeded with, followed by a retrocolic anastomosis, between the stomach and the jejunum. The abdomen was closed with tier sutures. The operative procedure was carried out under my supervision by the Resident in Surgery at Charity Hospital, Doctor Di Leo.

The patient recovered and left the hospital March 13, 1942.

Pathologic Report.—Clinical Diagnosis: Carcinoma of the duodenum with nodal metastasis. (M1634 S-42-1040).

"*Gross:* The specimen consists of the distal 13 cm. of the stomach and the proximal 7 cm. of the duodenum. The stomach appears normal except for a recent hemorrhagic mucosal ulceration measuring 1.5 cm. in diameter which is located 6 cm. from the pylorus. There is a rounded pedunculated mass located in the duodenum immediately beyond the pyloric ring, which measures 5 x 4 cm. and whose base to apex measurement is 2.5 cm. The diameter through the pedicle measures about 3.5 cm. The surface of the mass is somewhat irregular. The tissue is gray and granular and is quite friable. Along the greater curvature of the stomach near the pylorus there is a group of enlarged lymph nodes, which measures about 5 x 3 x 1.5 cm. On cut-section, these lymph nodes

show a complete loss of architecture, the tissue having been replaced by a homogeneous, gray, granular, somewhat friable tissue.

Microscopic: The tissue is composed of an infiltrating epithelial neoplasm which is arranged for the most part in solid cords and large sheets of cells. Some of the cords are only one or two cells in thickness. In a few areas there is definite evidence of acinus formation. The individual cells are quite large and contain an abundance of basophilic granular cytoplasm. The nuclei are rounded and contain one or more large nucleoli. Mitotic figures are not infrequently seen. The stroma is not abundant but is sufficient in quantity to separate the neoplasm in the masses previously described. Foci of inflamma-

FIG. 2.—Roentgenogram showing mucosal pattern changes.

tory cell infiltration are present throughout the neoplastic tissue. These consist largely of plasma cells. The enlarged lymph nodes show neoplastic tissue similar to that which was seen in the duodenum. *Pathologic Diagnosis:* Adenocarcinoma of the duodenum, with metastases to the regional lymph nodes." (Schenken)

The patient was readmitted to the hospital April 27, 1942, and died May 18, 1942.

Case 2.—T. R., white, male, age 64, was admitted to the hospital May 27, 1943, complaining of abdominal pain which he attributed to "liver trouble." He had previously had gastric disturbances which he had been told was due to "gallbladder trouble." At one time he reported that he had had some tarry stools.

Patient was obese. Skin was dry and scaly. There was no jaundice. Abdomen was symmetrically enlarged, there were no scars. Neither the liver nor the spleen were palpably enlarged. He did complain of pain on deep palpation in the right hypochondrium and along the course of the sigmoid.

A barium enema showed no evidence of disease of the colon. Gallbladder visualization was negative. G. I. series indicated: "Deformity of the duodenum, which suggests the possibility of an excavated ulcer." On the basis of this report the possibility of carcinoma of the duodenum was considered.

Since there was no evidence of jaundice it was our impression that if carcinoma of

the duodenum did exist, it must be suprapapillary. The patient was admitted to Touro Infirmary with a view of operating, and if possible of performing a radical resection. Further roentgenologic studies showed: "The stomach is of the 'J' type and lies high in the abdomen. The impression is obtained that the gastric walls are smooth and flexible and that peristalsis passes without interruption to the pylorus. No gross organic lesion can be demonstrated in the stomach. The duodenal bulb fills immediately and its contour is smooth and regular. *In the descending segment of the distal duodenum, just above the ampulla of Vater there is a sharply demarcated, large, oval filling defect,* measuring 4 x 3 cm. in the greatest projected diameter. The mass which produces it, apparently arises

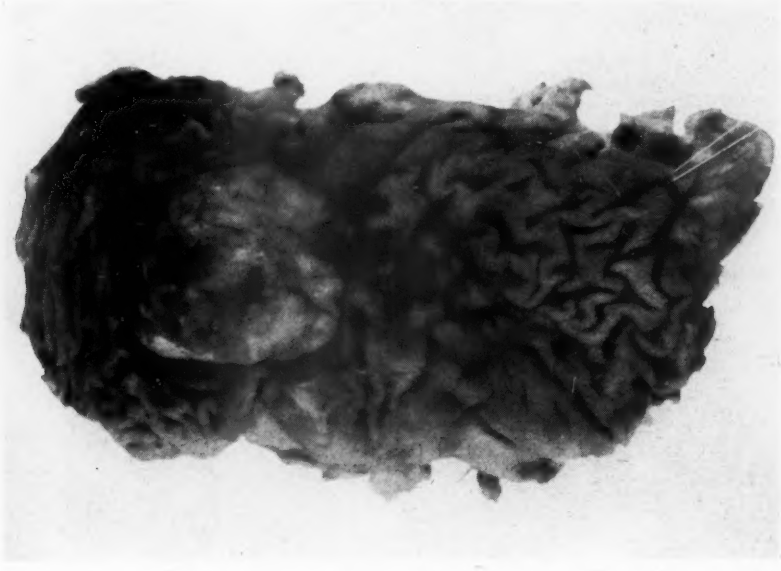

FIG. 3.—Gross appearance of lesion.

from, or deeply impresses, the lateral wall of the duodenum, the thin contrast stream outlining the medial border of the abnormal mass.

"The findings are suggestive of an intraluminal neoplasm, possibly benign. An ectopic pancreatic lobule (annular pancreas), might well produce the same deformity. The distal duodenum is normal. In six hours the stomach and duodenum are empty, the barium being distributed through the lower small intestine and proximal colon." (Teitlebaum)

Laboratory Data.—Red count 3,650,000. Total white count 8,250, neutrophiles 68 per cent, eosinophiles 2 per cent, lymphocytes 28 per cent, monocytes 2 per cent. Blood chemistry: Calcium 10.2 mg., phosphorus 2.88 mg., dextrose 87 mg., N.P.N. 30. Metabolism: Plus 6 to plus 1. Electrocardiographic findings were reported within normal limits.

Operation.—June 2, 1943: *Preoperative Diagnosis:* Carcinoma of the duodenum.

Under nupercaine spinal anesthesia, a long paramedian incision was made on the left side. The stomach was small and we were able to completely exteriorize it. The pylorus was easily identified and seemed perfectly normal. About two inches beyond the pylorus, and on the medial aspect of the duodenum, there was a thick infiltrated mass which was probably an inch and one-half long, easily palpable but not movable. The gallbladder was not adherent to the duodenum. It did not seem feasible to undertake a resection of the stomach and duodenum. The great amount of fat in the mesocolon obscured vascular markings. An anterior gastro-enterostomy was done. It was realized that this was only

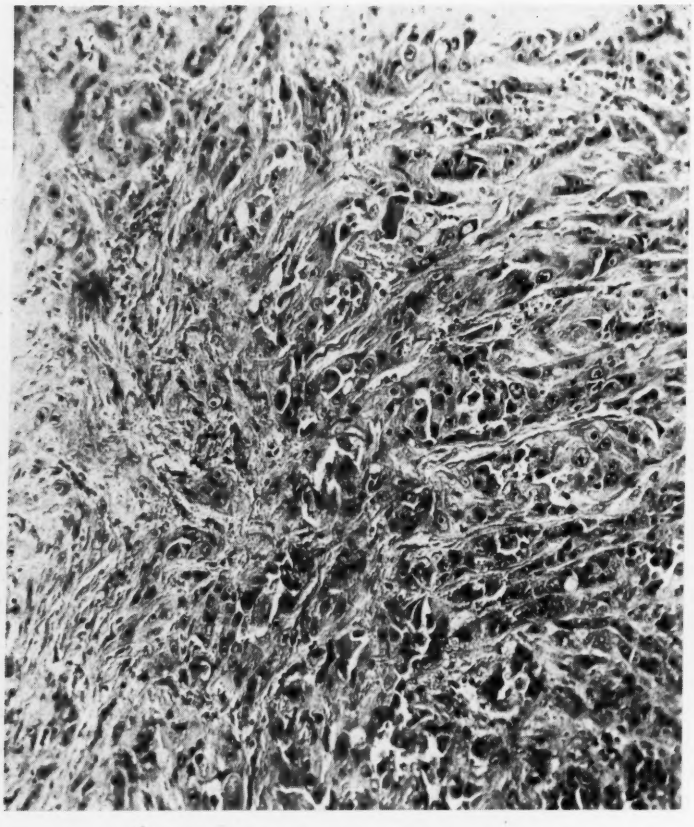

FIG. 4.—Microphotograph (see description in pathologist's report).

a palliative operation, but we felt that any attempt to be radical might result in a tragedy. The abdomen was closed.

Following the operation the patient persisted in vomiting and, on June 16, he was reoperated because of a vicious circle following the gastro-enterostomy. At this time, we found that he had an intestinal obstruction due to adhesions, an entero-anastomosis was done. The patient improved for a few days, but later developed a lower obstruction and, in spite of every effort, expired on July 7.

Much of the enthusiasm which I experienced because of a correct diagnosis, was dampened because of the failure of our efforts to accomplish anything for the patient. Investigation of the literature does not do a great deal to warrant optimism in regard to carcinoma of the duodenum; particularly, of the suprapapillary portion.

It is admitted that carcinoma of the duodenum is a rare disease. Compiled statistics indicate that: (1) It is found in 0.03 per cent of all carcinomas. (2) Carcinoma of the duodenum constitutes 0.3 per cent of all cancers involving the intestines. (3) One-half of the carcinomas of the small intestines are to be found in the duodenum.

It will not be my purpose to compile statistics, nor will I review the various

FIG. 5.—Mr. T. R. Note change in mucosal pattern of duodenum.

theories which had been advanced. A large number of articles have dealt exhaustively with the subject of carcinoma of the duodenum. During recent years Hoffman and Pack, Stewart and Lieber, and Duff, Foster, and Bryan, have capably presented the subject.

The diagnosis of carcinoma of the duodenum preoperatively, according to the literature, has been made only infrequently. This is depressing in spite of the fact, that a rather careful, clear, clinical picture has been recorded for nearly half a century. Throughout the literature one finds such statements as: "Until recently the diagnosis has been rarely made during life or before operation, and even with the aid of roentgenology, it is often very difficult to recognize the disease."

The insidious character of the disease and the gradual development of an obstruction, with symptoms common to those of carcinoma of the pylorus, with obstruction, may exist for a period of from six months to four years, without a correct diagnosis being made.

Great progress has been made as a result of the use of roentgenology and

FIG. 6.—Mr. T. R. Note change in mucosal pattern of duodenum.

gastroscopic methods in intrinsic lesions of the stomach and other portions of the bowel, but apparently similar positive progress has not been made in conjunction with carcinoma of the suprapapillary portion of the duodenum. Thus, Stewart and Lieber state: "Roentgenographic studies are of little direct value in making a positive diagnosis of carcinoma of the suprapapillary portion of the duodenum." These same authors do not think it is possible to distinguish defects in the suprapapillary segment due to carcinoma from deformities due to ulcer.

Hoffman and Pack, in 1937, concluded that: "A diagnosis of cancer of the duodenum is attended with so much difficulty that most of the recorded cases are recognized only at operation or at autopsy" and further state: "In only a few cases reported in the literature has the condition even been suspected. Because of its rarity it is usually not even considered as a possibility in a differential diagnosis. If *the possibility of carcinoma is given consideration,*

it should be feasible to make a presumptive diagnosis in certain of these cases." Relative to the roentgenologic findings they conclude that they "are usually interpreted as deformities and contractures due to duodenal ulcer."

A review of the literature and a summary of the experience induces a very discouraging picture. Unfortunately, the signs and symptoms on which, up to the present time, even a suspicion of the real nature of the lesion are, in reality, the manifestations of an obstructive stage of the disease. This is of necessity, a very late stage and one in which hope for anything like good end-results, so far as cure or prolongation of life are concerned, are extremely bad.

CONCLUSIONS

1. One cannot help but be impressed with the insidious character of carcinoma of the duodenum, particularly the suprapapillary type, as these cases are not associated with jaundice.

2. The almost asymptomatic course of the disease until it becomes obstructive in character is important.

3. The absence of pain until the late stage of the disease, seems to be an outstanding characteristic.

4. In spite of the fact that so many insist that roentgenologic manifestations are not of diagnostic value, I do not believe that anyone could doubt for a moment the importance which should be attached to such mucosal pattern changes as were noted in the cases which are herewith presented.

5. Unfortunately, the extensive nature of the disease which was found in one of our cases, made it impossible to do more than palliative procedures. In the other instance, it was possible to perform a resection of the stomach and portion of the head of the pancreas and duodenum, followed by a Pólya-type of operation, but this only prolonged the patient's life for a period of two or three months.

6. The importance of being conscious of the possibility of carcinoma of the first portion of the duodenum, cannot be overestimated if eventually our results from a surgical standpoint, are to be improved.

7. Since the differentiation between ulcers of the duodenum and carcinoma of the duodenum is so difficult to make, it becomes necessary to revise our attitude in regard to some duodenal ulcers in individuals past middle life.

8. If carcinoma of the duodenum is to be cured operation must be undertaken before the obstructive stage, which, in reality, is an antemortem manifestation. Such an admission is a sad confession of one's shortcomings.

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DISCUSSION.—DR. DERYL HART, Durham, N. C.: In view of the rarity of carcinoma of the duodenum as distinct from carcinomas of the head of the pancreas and the ampulla of Vater, I want to call attention to the possibility of aberrant pancreatic tissue being confused with carcinoma both in the roentgenograms and at the operation.

I have seen a number of patients with aberrant pancreas in the wall of the duodenum, and would like to call attention to two recent instances. The first had an annular mass of pancreas surrounding the duodenum, and was asymptomatic until a relatively mild attack of pancreatitis caused sufficient swelling to produce partial duodenal obstruction. At the time of operation the presence of the pancreatitis was an aid in making the diagnosis. The obstruction was relieved by an anterior gastro-enterostomy.

The second patient presented much greater difficulties in diagnosis. In a man of advancing years, with a progressively increasing obstructive jaundice (van den Bergh test (direct), bilirubin 29.4 mg. per 100 cc. before operation) roentgenograms of the barium-filled duodenum revealed a filling defect at a point interpreted as the region of the ampulla of Vater. A diagnosis of carcinoma of the ampulla of Vater was made and operation was performed, with the object of resecting the tumor with a portion of the duodenum. At operation, the first indication of an error in diagnosis was the presence of a nondistended gallbladder; further examination revealed a small common duct. On palpation, a mass, measuring 1 x 1.5 x 2 cm., was felt in the duodenum in the region of the ampulla, as had been demonstrated roentgenologically. The duodenum was opened; the lesion had the appearance of the ampulla of Vater with malignant change and an ulceration of 5–8 mm. in diameter. However, the ampulla was located about 1.5 cm. from this mass. The portion of the mass protruding into the duodenum with the ulceration was excised, and microscopic study revealed normal pancreas, with an ulcer overlying the point of maximal protrusion into the duodenum. The duodenum was closed, the patient developed signs of liver insufficiency, and died four days after operation.

Such conditions require that we keep in mind the possibility of benign lesions being confused with carcinoma, with resultant extensive resections carrying a relatively high risk.

CONGENITAL PYLORIC STENOSIS*

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CONGENITAL PYLORIC STENOSIS is the most common condition that requires surgical treatment in the first few weeks of life. The baby with this condition has an increase in size of the pyloric muscle, so that the lumen is compressed and obstructed and there are signs of high intestinal obstruction.

The mortality in this condition varied from 50 to 75 per cent prior to 1908. Today, proper surgical technic combined with good preoperative and postoperative care has reduced the mortality in competent hands from 1 to 2 per cent.

Fredet, in 1908, devised and performed something like the operation finally developed by Rammstedt in 1912. This has been aptly named "pyloromyotomy," or the Fredet-Weber-Rammstedt operation, or the Rammstedt operation. Fredet performed his first operation October 12, 1907, by carrying his longitudinal incision only as far as the submucosa, leaving the stomach unopened. He then sutured the muscle transversely. In September, 1911, Rammstedt operated following Fredet's and C. Weber's operation, which was similar, and was impressed by the ease by which the edges of the wound in the muscular wall of the pylorus gaped and the submucosa bulged. The patient continued to vomit for a few days and suddenly ceased and thereafter made an excellent recovery. He remembered that the muscle edges had been tight and closed under tension and that the sutures must have given way and the wound righted its direction, allowing the edges to separate and the mucosa to protrude and thus the obstruction was overcome, so, on January 18, 1912, he tried this idea and the patient recovered without incident. The same year he reported his two operative cases, setting forth very clearly his method and advocating the technic as best suited to deal with the problem of congenital pyloric stenosis. Rammstedt advocated the use of the term "pylorotomy," but it has generally become known as the Rammstedt operation, or "pyloromyotomy." Infantile pyloric stenosis seems to me the best name for the condition.

The scattered accounts of cases closely corresponding to congenital pyloric stenosis were published as early as the seventeenth century and the case of Hildanus, a famous German pediatrician, seems to be the first, in 1646. Blair discussed a case in 1717; C. Weber, in 1758, and Armstrong, in 1777, described a typical case in an "Account of Diseases Incident to Children from their Birth to the Age of Puberty: With the Successful Method of Treating them."

An infant had died of "watery gripes" at the age of three weeks, Armstrong notes: "The extremities felt very cold, except when kept warm by art.

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The face, likewise, felt cold and looked very pale, pinched and distressed." The pathologic picture is set forth in considerable detail: Most of the stomach, including the whole of the fundus, was in a "tender state," as if "the coats had been reduced by maceration to almost gelatinous consistence," but toward the pylorus the stomach structure was "firm enough." The stomach was quite distended with curdled milk and victuals with which the nurse had crammed the child, but the whole intestines were remarkably empty." From these findings Armstrong attempts to find an explanation of the condition. "There was no morbid appearance to be observed anywhere but in the stomach and this viscus being so full while the intestines were almost empty, it looked as if the disease had been chiefly owing to a spasm in the pylorus which prevented the contents of the stomach from passing into the duodenum." He comments that: "Perhaps cases of this kind are more frequent than is commonly imagined," and adds: "What is remarkable, this was the third child (and they never have had any more) which the parents have lost at the same age, and with the same disease."

The first case of pyloric stenosis reported in this country is credited to Hezekiah Beardsley, who, in 1788, presented a "Case of Scirrhus in Pylorus of an Infant" before the Medical Society of New Haven. It appeared in 1788, in the first Medical Periodical published in the United States. It was discovered and made public again by Sir William Osler; and for many years it was believed to be the earliest example of the disease then extant. In the early nineteenth century Pauli (1839) and Williamson (1841) made accurate independent observations of this condition, and Dawosky (1842) for the first time described the projectile character of the vomiting.

In 1887, at the Wiesbaden meeting of the Gesellschaft fur Kindeenheilkunde, Hirschsprung described two cases of congenital pyloric stenosis. He gave an excellent account of the gross pathologic changes and expressed the supposition that these alterations were the cause of the definite clinical form of infantile vomiting. Within a brief time after Hirschsprung's presentation the disease became widely known.

I have reviewed the transactions of the Southern Surgical Association on this subject.

First, there is a paper by Dr. G. C. Gavin, of Mobile, Alabama, presented at the Twenty-fourth Annual Meeting, held at Washington, D. C., in 1911. He gives a very interesting discussion and report of a case, male, age seven weeks. It was diagnosed and he operated upon it, performing a gastro-enterostomy in September, 1911. He had good illustrations and drawings of the case. The patient made a good recovery.

Next was a paper by Dr. James F. Mitchell, of Washington, read at the Atlanta Meeting, in 1913. He opens his paper by saying: "On first thought, it would seem that an apology is due the association for presenting such a well-worn subject." He gives a most interesting discussion about the theory of the etiology of the disease and has a number of good illustrations of the cases. He reports three cases. A gastro-enterostomy was performed on each

one of them, and all of the patients recovered. He mentions Rammstedt's operation in his paper but did not employ it on these three cases. Doctor Mitchell says: "Dr. Dean Lewis, of Chicago, had four successful cases." The paper was discussed by Dr. Charles Mixter, Jr., of Boston, and Dr. C. Coffey, of Portland, Oregon. Both gave very interesting discussions. Doctor Coffey greatly laments the fact that the cases were not brought to the surgeon soon enough and that the medical men should be given further education; and Doctor Mitchell finishes his paper by saying: "That the chances for the recovery of these patients depends on getting them to the surgeon early."

Next was a paper by Dr. William A. Downes, of New York, read at the Cincinnati Meeting in 1915: He first reported in April, 1914, 22 cases, and in 1915, 44 additional cases—66 personal cases in five and one-half years. He gives a very full and complete discussion of the cases, the symptoms, and causes of death. His cardinal symptoms were: Projectile vomiting; tumor; peristaltic waves; gastric distention; rapid loss of weight; and marked constipation. Nearly half of these 66 cases had a gastro-enterostomy. He first incised the circular muscle, after the Rammstedt method, in October, 1914. In 19 cases he first employed the Rammstedt procedure, after which he made a small opening in the anterior wall of the stomach and passed a sound into the stomach and through the pylorus, making sure that the pylorus was patent, but he decided that these cases did no better than the ones in whom a gastro-enterostomy had been performed, so he quit that and employed the Rammstedt procedure in the last 34 cases. His mortality in the 66 cases was about 23 per cent. Of course, many of his cases were moribund when they were admitted. The preoperative preparation was not emphasized. He advocated beginning the incision at the stomach end and mentioned the dangers of getting into the duodenum. In his conclusions he brings out the advantages of the old operation and the new Rammstedt procedure. The gastro-enterostomy is more difficult and takes longer to perform. The Rammstedt procedure is simple, easy and requires less surgical skill and the obstruction is permanently removed and the normal continuity of the intestine throughout is preserved; that the method is open to criticism in that it leaves an uncovered wound, and the abdominal cavity is protected from contamination only by a thin layer of mucous membrane, and as the scar contracts the obstruction will reform; and he recommends that a gastro-enterostomy be performed if in any case there is reason to suspect that the lumen of the pylorus does not become patent after division of the circular muscle, and he says: "That continued experience and longer observation of the cases may bring out more serious objections to partial pyloroplasty, but until some better method is proposed the method according to Rammstedt should be the one of choice." "The success or failure of the operative procedure is determined in a large measure by the length of time elapsing between the onset of symptoms and the time of operation."

Next, at the Fortieth Annual Meeting at Augusta, Georgia, in 1927, a paper on "Congenital Pyloric Stenosis" was read by Dr. J. M. Mason, of

Birmingham, Alabama. He gives a most excellent presentation of the subject. He reviews the literature before that time and pays tribute to Dr. William A. Downes, whom I have quoted above. He quotes from Wollstein: "That the wound in the pylorus is healed within nine days. The pylorus has returned to normal size within a month. The gap between the cut ends of the muscle has practically disappeared in six weeks. In two years only a thin line of connective tissue fibers separates the two muscle ends and the stomach is quite normal. Thus, in contrast to gastro-enterostomy, which leaves the pylorus unchanged, the Rammstedt operation cures the pyloric lesion." He contrasts the difference between the medical and surgical treatment and recommends that all cases should be sent to the surgeon and operated upon, and he emphasizes the preoperative and postoperative care. He believes in suturing omentum over the pyloric wound. He gives a most excellent description of the operation and incident dangers, and reports 25 cases, seven of which he personally operated upon.

There has been no mention of congenital pyloric stenosis in the Transactions of this Association since Doctor Mason's paper, in 1927. The whole history of congenital pyloric stenosis is covered by these papers which are in the records of this Association, and I feel that we, as Fellows, should be very proud of all of these men who have done this most excellent pioneer work.

Another paper which I reviewed on congenital pyloric stenosis was published in February, 1931, in the Tennessee Journal of the State Medical Association, and was written by our distinguished friend, and president of the Association, Dr. Barney Brooks. He gives a most interesting and exhaustive presentation, with report of ten cases, and many practical points in the diagnosis and treatment. His experience covered the period of his connection with the St. Louis Children's Hospital, and the Vanderbilt University Hospital. He says "that in the early period gastro-enterostomy was accepted as the operation of choice."

The sign, which in his opinion, far outweighs in importance any point in the clinical history or any physical sign, is the ability to palpate in the region of the pylorus a small, firm, hard tumor. He believes this sign to be essential in making the diagnosis of congenital pyloric stenosis. He then says, "the ability to palpate this tumor must be a sort of 'knack' because he has frequently been able to feel this tumor and at the same time he has not been able to demonstrate its presence to any of his assistants." The method he found useful is as follows: "Give the child a bottle at the same time the examining hand is resting upon the infant's abdomen. During the very brief time in which the child first takes the bottle, the abdomen may be sufficiently relaxed to feel the pyloric tumor. Any one having once felt the tumor characteristic of congenital pyloric stenosis can never fail to recognize it again, even if the fingers touch it for only a fraction of a second."

He believes that the examination of the gastro-intestinal tract by means of a barium meal and roentgenology is of value only when its limitations are appreciated. If barium is put into the stomach and some of it is seen to pass

readily into the duodenum, pyloric obstruction is obviously not present. In a great many instances it has been found that the barium failed to pass into the duodenum in instances of infants without congenital hypertrophic pyloric stenosis. These instances were those in which the barium was vomited very soon after being taken, or were the result of true pylorospasm. He is inclined to believe that roentgenologic examination with the barium meal may be very good evidence against the existence of a congenital hypertrophic pyloric stenosis, but that it could never be conclusive evidence of its presence. In a statement he recently made to me he says: "Unless the case is clinically certain always give the child a little barium and examine fluoroscopically. Only last week this procedure kept me from operating upon a first born male who was vomiting and who had a palpable tumor in what I thought was the right place. The fluoroscope showed the pylorus wide open and the palpable tumor, a lymph node, I suppose, quite separate from the duodenum." He believes in the use of local anesthesia for the operation, and makes his incision just above the liver edge. "The liver then acts as a perfect stopper and the baby can not eviscerate, no matter how hard he strains." He believes that the pyloric tumor should always be divided from left to right, and uses the handle of his knife as a chisel rather than the cutting edge. In his recent communication he says "that he has done now well over 100 cases without a death and often takes two or three days getting the baby ready."

I feel that these papers of Doctors Gavin, Mitchell, Downes, Mason and Brooks on this subject are outstanding contributions, and I am sure all of you Fellows present will agree with me.

Etiology.—The cause of congenital pyloric stenosis is not understood. There are a number of theories which attempt to explain it. It seems to me that three factors enter into its formation. Congenital hypertrophy of the pyloric muscle; spasm, which may be secondary to the congenital hypertrophy; and an added compensatory or work hypertrophy. As far as I am concerned, the etiology of this condition is far overshadowed by practical consideration of the surgical treatment. It affects male children predominantly, in a ratio of about nine or ten to one. There seems to be a preference for first born babies who are robust and above the average weight at birth. From the published cases it would appear that the parents are most often young, healthy and in favorable economic circumstances. Familial occurrence, noted by Armstrong in 1777, has been frequently mentioned in later reports. Three cases of pyloric stenosis in the family of five children has been seen. The occurrence of pyloric stenosis in parent and offspring has been observed more rarely.

Pathology.—The pathologic findings are hypertrophy of the circular pyloric muscle, and an actual increase in the number of smooth muscle fibers. This produces an olive-sized, bulbous or fusiform mass which has a smooth external surface. It seems almost as hard as cartilage. The lumen of the stomach is gradually reduced in size toward the pylorus, but the lumen of the duodenum assumes its full size at once, due to the abrupt termination of the pyloric sphincter at its distal end.

Symptoms.—The symptoms of all cases are those of high obstruction and severe loss of body fluids. Doctor Ladd says "that if the five cardinal symptoms are present, namely, scanty stools, projectile vomiting, loss of weight and dehydration, visible peristalsis and palpable tumor, roentgenologic examination is not necessary nor desirable." The symptoms usually start within three weeks and the initial symptom is vomiting, which is projectile in character and seldom occurs before the ninth or tenth day after birth and usually before the child is 12 weeks old. At first, the vomiting may be little more than just regurgitation but soon it becomes more forceful and projectile in type. The vomitus does not contain bile but only gastric contents. Each feeding may be returned immediately but if the stomach is dilated several feedings may return together. Since little of the food really passes the pylorus there is constipation. Diarrhea sometimes occurs as the result of infection or starvation and the passage of accumulated intestinal juices. The child is continually hungry because so little food passes into the intestines to be absorbed. Therefore, he will eagerly nurse or take the bottle. Loss of weight is rapid. According to the degree of stomach peristalsis, waves are visible passing from left to right. These should be looked for immediately after feeding while the stomach is under maximum stimulus to overcome the obstruction.

The pathognomonic symptom which makes certain the diagnosis of congenital pyloric stenosis is the presence of a palpable tumor in the right upper quadrant, a little above the level of the umbilicus at the outer border of the right rectus muscle. One observer has found its location by watching for the point where the peristaltic wave ends. But the stomach should be empty for palpating this mass which is olive-shaped and indurated. Doctor Ladd says "the pyloric tumor has been felt in 95 per cent of all his cases. The amount of fecal residue is decreased as the pyloric obstruction is increased, therefore, the stools become less frequent and scant. With continued starvation, failure to gain weight or loss of weight always occurs. Dehydration is evidenced by wrinkles appearing in the skin anywhere on the body. He looks just like a little old man."

Physical Findings.—Examination of the abdomen is of the utmost importance. There may be some distention in the area of the stomach and palpation may show a distended stomach if the baby has recently nursed. Gastric peristaltic waves can be seen passing from left to the right. Palpation of the right upper quadrant with tips of the fingers will reveal, in most cases, the pyloric tumor.

Roentgenologic Examination.—Roentgenologic examination is not always necessary. There are, however, a few cases in which the history is atypical, or the physical examination is unsatisfactory, and whenever the diagnosis is in doubt it is best to obtain additional information by roentgenologic examination. A film of the abdomen without barium will often show a large, dilated, gas-filled stomach, with relatively little gas in the intestine beyond the pylorus. If this is not satisfactory, a routine gastro-intestinal examination may be

done with a little barium introduced in the milk formula or given in a little water, which will demonstrate an enlarged stomach with a rounded pylorus and shows a ballooning forward and to the right, and greatly increase peristaltic activity and the passage of little or no barium into the duodenum. Films taken later will indicate how much retention is present, and a retention of a large portion of the barium at this time shows a pyloric obstruction.

Preoperative Care.—Administration of parenteral fluids is the most important factor in improving the condition of these dehydrated patients. Fluids should be administered night and morning of each preoperative day. Intravenous 10 per cent dextrose in sterile water and salt solution by hypodermoclysis and blood transfusions are of great help. For the poorer risks oral feeding should be continued with the preoperative care. Vitamin C has been given by some of the observers.

Operative Procedure.—Operation for congenital pyloric stenosis is not an emergency procedure, and a baby destined to surgery should be prepared for operation by the medical regimen outlined above. Generally speaking, the worse the infant's condition, the earlier one should attempt operative relief, but plenty of time should still be taken to get the baby in as good condition as possible.

Ether is administered by the drop open cone or mask method. I have used this method in all of the cases I have operated upon and have found it very satisfactory. In my experience babies have taken ether better than any other anesthetic, so I have continued to use it in all cases.

For skin preparation I usually wash with soap and water followed by ether and a weak solution of iodine. If the stomach seems to contain much fluid I sometimes pass a catheter through the nose and into the stomach and wash out with sterile water and leave the catheter in place during the operative procedure.

I make a high right rectus incision beginning above the edge of the liver and extending down an inch and a half or two inches. When the abdomen is opened the right lobe of the liver is retracted upward and the pylorus is delivered into the wound and the tumor held between the thumb and index finger and a longitudinal incision is made over the tumor at its least vascular part until the mucosa is bulging. Then the back of the knife is used to complete the operation until the mucosa is bulging into the wound. Any bleeding can usually be controlled by moist pads. If that does not control the bleeding the vessels should be ligated with fine silk. If there is considerable bleeding a strip of muscle from the rectus may be sutured in the pyloric incision. It is most important to see that all bleeding is stopped before the abdominal wall incision is closed because death has been reported due to hemorrhage from the pyloric incision. The complication most to be dreaded is accidental opening of the duodenum. The change from thick pyloric tumor to thin duodenum is abrupt, so great care must be exercised in separating the cut edges of the muscle toward the duodenal end. I usually do not attempt to cover the pyloric defect with omentum or serosa. When I am satisfied about

the wound the pylorus is then dropped back into the abdomen. Then the wound is closed in layers. I use fine, chromic catgut making as few knots as possible, suturing the peritoneum and muscle and fascia using the same strand, and then close the skin with a few interrupted silk sutures. I have never used any abdominal wall incision but the one described. There is in use the gridiron incision, and several others, but I have had no experience with them. The head of the bed is lowered until the baby recovers from the anesthetic in order to prevent aspiration of mucus.

Postoperative Care.—After pyloromyotomy for congenital pyloric stenosis gastric peristalsis is inhibited for several hours and is frequently depressed for longer than that. In average cases evacuation from the stomach through the pylorus of material ingested soon after operation does not begin until about eight hours and is not complete until 24 hours after operation, therefore, I do not believe in the early and frequent administration of water and food during the first postoperative day. Water is given sparingly at first and if the child is not vomiting at the end of 12 hours a suitable formula, or breast milk, is started and given every three hours. Most cases vomit a little for two or three days. Parenteral fluids are given in an adequate amount for the first two or three days and blood transfusions whenever necessary. At the end of two weeks, generous feedings are tolerated, the baby is gaining weight normally, and the wound has healed, and the skin sutures are out. The operation restores the stomach to normal and the child grows up to normal gastric functioning.

I have reviewed the case histories of 27 babies which I have operated upon since 1920. These were all private cases. I have used ether anesthesia, and have performed the Rammstedt pyloromyotomy through a high right rectus incision in every case. In most cases the baby was one to two months of age when it came to operation. There was one death. In this case there was no bleeding and no perforation of the duodenum. The patient seemed to be doing all right several hours after operation and suddenly he became worse and died. No autopsy was obtained. In the remaining 26 cases the operations were successful and the babies recovered. There were two females and 25 males. Nearly all of these cases were referred to me by one pediatrician, Dr. Thomas M. Marks, of Lexington, Kentucky. I have had the advantage of his assistance in the diagnosis and preoperative and postoperative care and I feel that the success obtained is largely due to him.

SUMMARY

Congenital pyloric stenosis is seen most often in male infants who were robust at birth. There is usually an interim of a few days or weeks during which the infant thrives. Symptoms start suddenly with vomiting which becomes projectile and is followed by constipation, loss of weight and exaggerated gastric peristalsis. Careful search will reveal a palpable mass, representing the hypertrophied pylorus, in most cases. The treatment of choice is Rammstedt's operation (pyloromyotomy). Preoperative treatment is most

important. The results of operation are excellent in the great majority of cases. Twenty-seven cases were briefly discussed.

One writer sums up the whole situation when he says the criteria of success are threefold: Early recognition of the pathology; prompt operation by an experienced surgeon; and careful postoperative management by a competent pediatrician. Indeed, the pediatrician holds the key to the outcome of any case. Brooks says in the long run the responsibility for the child's salvation rests upon him, for anyone who can even remotely be considered a surgeon can perform the operation successfully.

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DISCUSSION.—DR. DERYL HART, Durham, N. C.: I enjoyed Doctor Vance's presentation very much. Most people agree that this condition is best treated surgically. In a series of more than 60 patients so treated, over a period of 16 years, we have had no fatality, and in only a few cases has the duodenum been opened.

These results depend on close cooperation between the pediatrician and the surgeon and a simplified technic, rather than on individual operative perfection, since many of the operations have been performed by the changing surgical residents. Our technic differs only slightly from that of Doctor Vance. All operations have been performed under local anesthesia through a high right rectus incision, lying predominantly over the liver. The lower edge of the liver is retracted upward so the pyloric tumor can be delivered. This is grasped between the thumb and index finger of the left hand, and a longitudinal incision is made in the anterior relatively avascular area, extending through the peritoneum and superficial muscles from the duodenum up onto the stomach above the tumor. No further cutting is done, the remaining circular muscular fibers being broken across by tension. This is accomplished by inserting the point of a hemostatic forceps between the edges of the peritoneal incision and spreading very slowly and carefully. The instrument is held tangential rather than perpendicular to the pyloric tumor so as not to run any chance of the point perforating the lumen. As the muscle fibers break, the submucosa is exposed and, with further spreading, it bulges up into the incision as the muscle retracts and separates from the submucosa.

The advantages of this procedure in our hands is that no instrument ever touches the submucosa, the amount of tension can be accurately controlled, and the entire procedure is under visual control. The only possible chance of entering the lumen is at the duodenal end, where too great tension can tear through the more delicate structures. This muscular division should be carried down to, but not onto, the thin-walled duodenum. After completing the division and controlling any bleeding, the raw surface may or may not be covered over by a free or pedicle graft of omentum. The liver then is allowed to fall back in place to underlie the incision, thus giving some protection against evisceration. The peritoneum is then closed with a continuous suture of catgut, or interrupted sutures of silk or cotton. The remainder of the incision is closed in layers with interrupted sutures of fine silk or cotton. Following such a closure, evisceration is extremely rare. In the rare cases where the lumen has been entered, the opening has been closed and convalescence has been satisfactory. The most amazing complication from this has been the delay in resumption of feedings in these already undernourished infants. There is always, of course, the fear of leakage and the development of generalized peritonitis.

DR. JAMES M. MASON, Birmingham, Ala.: This is a very interesting subject and I have been pleased with the manner in which it was presented. As in all radical procedures, the surgeon cannot get very far until he has the hearty endorsement of his medical colleagues. Going back into the history of the operative treatment of hypertrophic pyloric stenosis in children, we find that the Fredet-Rammstedt operation was first performed in 1911. William A. Downes, a former member of this Association, became interested in it and had operated upon 22 cases by 1914. In 1917, Holt, the leading pediatrician of his day, gave the operation his hearty approval in a paper which appeared in the Journal of the American Medical Association in that year.

Since that time the value of the operation has been definitely established, and the diagnostic signs and symptoms have become so well recognized that prolonged medical treatment is giving way to prompt surgical intervention. At the Children's Hospital

the pediatricians no longer persist in prolonged administration of atropine, or resort to thick feedings in infants who come in with a history of projectile vomiting, peristaltic waves, and with roentgenologic findings of prolonged barium retention in the stomach. When these conditions obtain, the Rammstedt operation is undertaken without delay and results have been uniformly satisfactory. The tumor is always sought for and, with experience, one can usually find it. Operation is not withheld, however, if the tumor has not been found and if the other symptoms are present.

In spite of the employment of the best technic, perforation of the duodenum may occur. In my earlier operations I had two deaths from this cause. Occasionally, evisceration will take place when the right rectus incision is employed. A transverse incision is just as easily carried out and the danger from evisceration is greatly lessened.

Dr. Rorert L. Rhodes, Augusta, Ga.: I just want to add several remarks and report two particularly interesting cases. In the first place I want to emphasize that one should not be impressed by failure to find projectile vomiting. When a child has reached the stage of exhaustion he does not vomit, and often that is when you see him. The second thing is that you do not always have a mass that is easily palpable, as Doctor Vance brought out.

Recently I have seen two cases I want to record. Just a year ago a child was brought in, ten months of age, weighing five pounds. The diagnosis of pyloric stenosis had been made at the age of three or four weeks by the doctor who delivered the child, and he had advised operation. During the intervening time the parents had taken the child all over the State of Georgia looking for someone who would cure it without operation. Finally they returned to their own doctor, who sent them to me. There was no projectile vomiting, no palpable mass. We gave a little barium by way of confirmation, and if any portion of that half-ounce had passed through in 48 hours the roentgenogram failed to show it. After the usual preliminary building up with fluid, glucose and a little blood, we went in and found no pyloric muscle at all. A hair of your head would have represented the constricting band encircling that pylorus to the extent that you could not get to it, the stomach and duodenum bulging over the constriction. It was perfectly obvious that a Rammstedt operation could not be done; one could not reach the cicatricial band without opening the duodenum. So I decided that the best thing to do was a Horsley pyloroplasty. One could feel the knife as it nicked this band but could not see it even with the pylorus wide open. There was no evidence whatever of a pyloric muscle. The child made a good recovery, I am happy to say, and at the last report had gained 25 pounds since operation.

I had a typical case a couple of years ago in which the Rammstedt operation was attempted and, unfortunately, as Doctor Mason just said, we are going to nick the duodenal mucosa in spite of all precautions, and I immediately converted from the Rammstedt to the Horsley procedure and had no cause to regret it, since the result was excellent.

Dr. Roger G. Doughty, Columbia, S. C.: I have had the experience of getting into the duodenum, and in two instances we simply closed the pylorus and rolled over to a second avascular area and repeated the Rammstedt procedure at that point. It seemed to work very nicely, and it is a simple thing to do.

I was much interested in what Doctor Rhodes said about adhesions. Some years ago we reported a small group of cases of persistence of the anterior mesogastrium. I think this is a condition which has been confused with pyloric stenosis by pediatricians. There is one differentiating point; in pyloric stenosis they do not vomit bile; in persistence of the anterior mesogastrium they vomit bile early.

Dr. Charles A. Vance, Lexington, Ky. (closing): This subject was so interesting to me that I spent too much time with these five papers. In the written paper I have covered all the points brought out in discussion. Doctor Hart says he uses a spreader for the pyloric wound; others have used forceps or scissors. I saw one man operate who used too much force with Kelly forceps and spread the wound open from end-to-end into the stomach and duodenum. After that I quit using spreaders and have used the back of the knife and get just as good results, and I have not punctured into the duodenum and stomach. I was very much interested in hearing about Doctor Doughty's other incision. I had not heard of it being done before and shall remember it and use it when it becomes necessary.

NEUROFIBROMA OF THE SMALL INTESTINE*

REPORT OF CASE

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PORTSMOUTH, VA.

BENIGN NEOPLASMS of any kind are uncommon in the small intestine. Rankin and Newell[16] reported, in 1933, that they found only 35 cases in the files of the Mayo Clinic. Morison,[30] in 1941, stated that he had studied material from 13,139 patients and only 21 benign tumors of the small bowel were discovered. Heurtaux,[2] writing in the last years of the nineteenth century, could find but 29 authenticated cases in all previous medical literature. Of the various types of small intestinal tumors the fibromata would appear to be among the rarest. In Rankin and Newell's series only six fibromata were found. Morison found only one fibroma in his studies. While Heurtaux's list contains three fibromata, von Bruns,[5] in his treatise on surgery, published at Stuttgart in 1913, notes that Heisig, reporting a case of fibroma of the small intestine upon which he had operated in 1897, had been able to find no similar instance in the literature.

It is difficult from the early reports to decide which of the fibromata reported were of neurogenic origin. Twentieth century pathologists have differentiated neurofibroma from fibroma in general, but in earlier cases details are so often lacking that it is not always possible to identify the type of fibroma that is being described. Very probably the growths which formerly were called "fibroma" would today be designated by both clinician and pathologist as "neurofibroma."

Intestinal neurofibromatosis was recognized and described some years before the beginning of the present century. In February, 1897, Albert Branca,[1] an intern working in Pierre Marie's clinic, published an account of one of Marie's cases, including an elaborate pathologic study. The patient, an old alcoholic, was admitted to the hospital, well advanced in pulmonary tuberculosis. The cutaneous surface was scattered over with typical lesions of von Recklinghausen's disease. At autopsy the characteristic nerve lesions appeared in many of the internal organs, including the intestines.

Branca concluded that although neurofibromata had previously been described only as appearing on the external body surface, precisely similar lesions might be produced in the intestines, given the proper conditions for proliferation. A case clinically similar to Branca's was reported by Leriche[4] in 1911. The patient was tuberculous and manifestly suffering from von Recklinghausen's disease, but was operated upon because he showed clinical symptoms of a growth at the pylorus. The operation disclosed a "fibroma without nerve elements undergoing sarcomatous degeneration," and the his-

* Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

tologic examination presented a picture similar to those later offered by twentieth century pathologists as "neurofibrosarcoma."

Since the recognition of neurofibroma as a separate entity, there has been no uniformity of opinion as to the etiology and origin of these rare growths of the small intestine. King,[6] in 1917, stated that it was his belief that the fibrous tissue of the nerve sheath is the probable site of origin, citing the cases reported by Leriche, Branca and Marie and Couvelaire.[3] All of these cases, however, were of generalized neurofibromatosis, with many intestinal

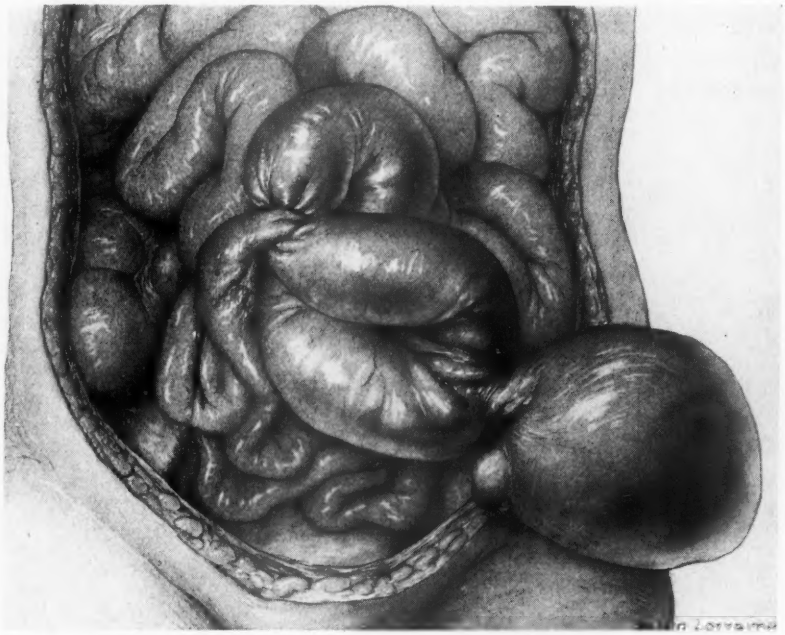

FIG. 1.—Sketch of operative findings showing the tumor twisted on its pedicle causing a volvulus.

tumors, which can only be regarded as histologically identical with the cutaneous neurofibromata, with which the patients were known to be affected. King adds that "an interesting point is that these tumors are frequently found in tuberculous subjects; also interesting is the fact that they are frequently associated with sarcoma."

The pathology of intestinal tumors arising in nerve elements seems to have advanced no further, either in continental Europe or North America during the decade which included the years of the first World War. A perusal of the literature of this period shows that there was much uncertainty and confusion in the classification of these neoplasms. In France they were variously termed "plexiform sarcoma" (Robert), "malignant leiomyoma" (Goullioud), "fusiform cell sarcoma" (Tedenat), "fibrosarcoma" (Bertrand of Montpelier), "fusiform sarcoma" (Goullioud), "fuso-sarcoma" (Walther) and "angiosarcoma" (Champy and Burty). An equal variety of designations

is to be found in American literature, and it may be added, an equal uncertainty in pathologic recognition.

The study of gliomata arising in various parts of the nervous system, especially in those of the viscera, presented by Lhermitte and Leroux[7] in 1923, cast some light upon the histology and etiology of these little understood neoplasia. They had been able to identify, in five cases, tumors situated in the wall of the digestive tract, the structure of which appeared in all respects to be identical with that of growths occurring in the peripheral nerves and in the spinal nerve roots. Four of these were at the pylorus or other parts of the wall of the gastric cavity, but one was "in the ileocecal region, and had developed in the thickness of the wall of the cecum, the mucosa of the intestine being normal."

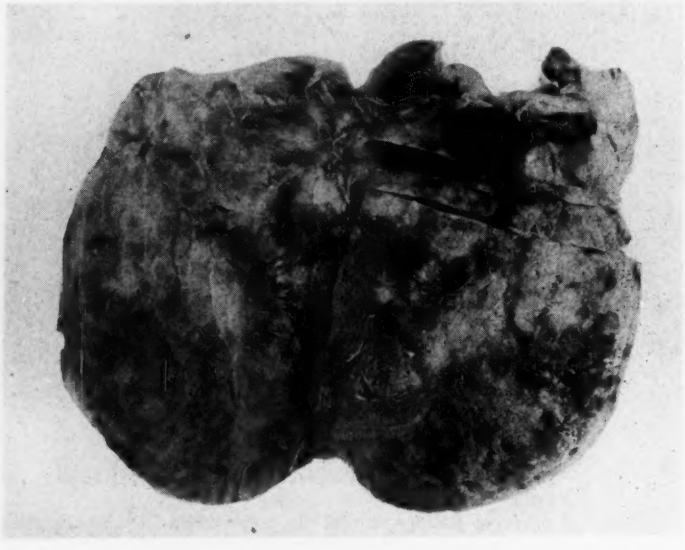

FIG. 2.—Photograph showing gross appearance of the interior of the tumor.

In 1924, Gosset, Bertrand and Loewy[8] published a paper in which they tabulated 66 cases of pedunculated tumors of the stomach, to which they added two cases of their own. The 66 cases had mostly been diagnosed as "malignant leiomyomas" or "sarcomas." They demonstrated by careful consideration of all pathologic material at their disposal (as described in the literature) that these growths were actually benign. Only six were assigned origins in nerve tissue and designated as gliomas, fibrogliomas or schwannomas. These neurogenic tumors were described as follows:

"A typical growth of the kind under discussion (that is pedicled tumors of the stomach) will prove under the microscope to have a structure identical with peripheral nerve tumors. Whorls of tissue appearing like connective tissue prove to be composed of fusiform cells, anastomosis between these whorls being very frequent. The structure of these whorls is very dense, and the individual whorls are often isolated in a stroma of connective tissue. This isolation of the whorls gives an appearance resembling cylindroma,

which is most often perceivable at the periphery of the growth, close to areas where degeneration is in progress. An appearance peculiarly characteristic of these growths is the nodule made up of cells in *palisade* formation, which is seen only in peripheral gliomas. Polycystic degeneration, at first limited to the protoplasmic zone near one of the nuclear poles of the individual cells, but later on forming large individual cysts, can be made out scattered through the entire structure of the growth. The elements deriving from the neurilemma (sheath of Schwann) do not proliferate by mitosis, but cleave longitudinally, so as to form a nodule with nuclei in line with the same axis. Because myxomatous degeneration is quite common, the swelling of the cell protoplasm, turning the cells into vesicles, so changes the appearance as viewed in the microscopic sections, that the growth is often mistaken for a connective tissue tumor, and its origin in the nerve sheath is wholly obscured."

These authors also cited a communication made by Paul Picquet, in 1922, to the Societe de Chirurgie, which was an anatomicoclinical study of a case

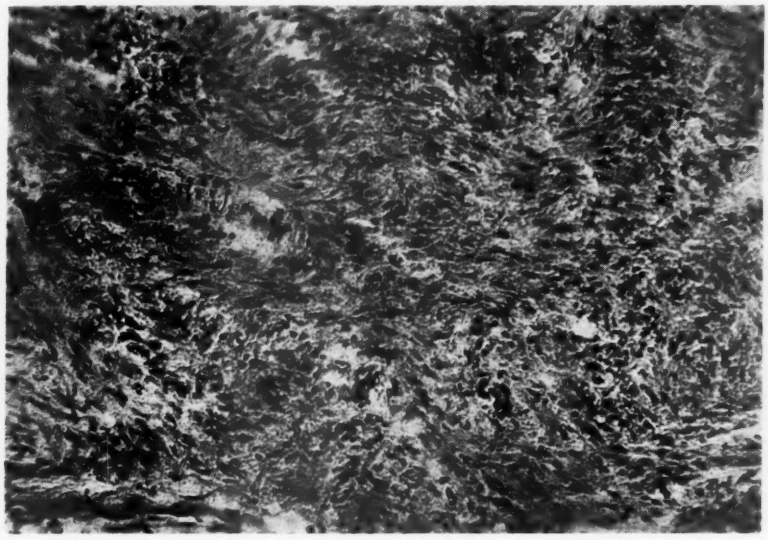

FIG. 3.—Photomicrograph (low power) showing oval shape of cells with frequent palisade arrangement of the nuclei.

of pedicled "exogastric" tumor. The histologic examination showed the growth to be of nervous origin, "analogous to the visceral tumors seen in von Recklinghausen's disease."

In 1932, appeared Masson's[14] monograph, Experimental and Spontaneous Schwannomas (Peripheral Gliomas), "addressed to that minority of pathologists who still refuse to admit that the fundamental constituent of the encapsulated tumors of the peripheral nerves is the schwannian synctium, a neurectodermal structure and not the mesodermal fibroblast." This did much to clarify the entire subject of tumors arising in the nerve sheaths, and to permit recognition of the benign nature of the majority of those growths when they appeared infrequently in the digestive tract. But Masson's opinion was not universally accepted. Four years later we find two French pathologists, Leveuf and Godard,[19] writing of "cavitary sarcomata of the small intestine in chil-

dren," and discussing, in particular, the pedicled variety, remarking: "These pedicled tumors are not true sarcomas; in the majority of cases they should not only be exempted from that classification, but should rather be definitely classed as neurofibromata."

Visceral neurofibromata may occur as a solitary growth or they may appear as multiple tumors. Ewing[27] states that they may be found anywhere in the intestinal tract "from the lips to the anus." The importance of establishing the true nature of these growths needs no emphasis. Although they may undergo malignant degeneration, they are no doubt primarily benign, and should be treated as such.

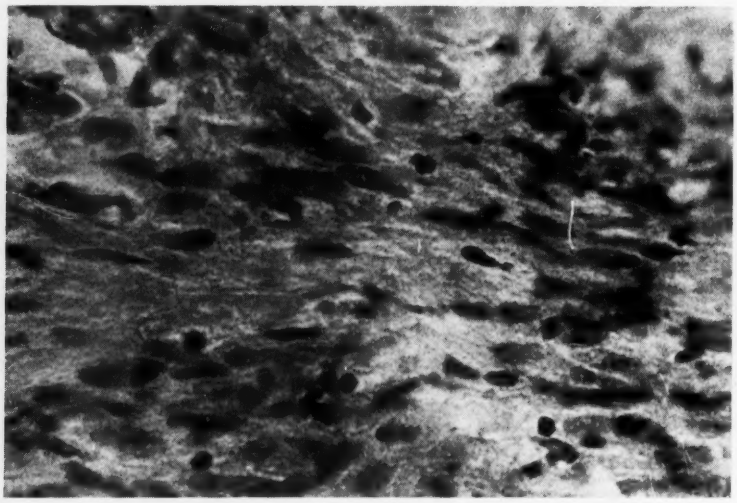

FIG. 4.—Photomicrograph (high power) of Figure 3 showing details of cell arrangement.

From a review of the more recent literature, it is evident that there is still a difference of opinion as to the classification and histogenesis of these tumors. They have been described under many different names. "Neurinoma" and "schwannoma" appear most frequently in foreign reports. They have also been designated as peripheral glioma, fibroblastoma, neurilemoma, perineural fibroblastoma and neurogenic fibrome, depending upon the opinion of the writer as to the structure or origin of the growth described. According to Ewing "a satisfactory classification of these tumors is difficult, owing to the complex structure of the nerve trunks, the uncertainty of the source of the supporting cells which accompany nerve fibers such as the cells of the sheath of Schwann, the neurilemma, the endoneurium and the lymph sheaths of nerve trunks, all of which participate in tumor growth."

On account of the confusion in terminology it has been extremely difficult to make an accurate estimate of the number of cases that have been reported. This has been particularly true in the preceding decade. However, I have been able to collect 18 cases that have been operated upon since 1929. From a study

TABLE I

ANALYSIS OF 18 CASES OF NEUROFIBROMA OF THE SMALL INTESTINE REPORTED SINCE 1929

Authors	Sex	Age	Principal Signs and Symptoms	Path. Report	Operation and results
Dudley (1930)[9]	F	49	Abdominal pain	Benign	Resection—Recovery
Picard (1932)[12]	F	54	Intestinal obstruction	Benign	Resection—Recovery
König (1932)[10]	F	40	Hemorrhage, acute peritonitis. Tumor felt per vagina	Benign	Resection—Recovery
Denecke (1932)[13]	F	53	Abdominal pain and palpable tumor	Malignant	Exploratory Celiotomy. Death within 10 days
Lemonnier & Peycelon (1933)[15]	M	42	Intestinal hemorrhage	Benign	Resection. Death from shock
Nordlander (1933)[11]	M	73	Intestinal hemorrhage	Malignant	Resection. Death several hours later
Baumeister (1934)[17]	F	59	Symptoms of acute appendicitis	Benign	Resection—Recovery
Borras & Benito (1936)[18]	F	54	Abdominal pain and palpable tumor	Benign	Resection—Recovery
Arnvig (1937)[20]	M	47	Intestinal hemorrhage—palpable tumor	Benign	Resection—Recovery
Bergendal & Sjövall (1937)[21]	M	29	Acute peritonitis	Malignant	Resection — Death from recurrence 4 yrs. later
Graffin (1939)[23]	F	51	Intestinal obstruction	Benign	Resection—Recovery
Babbini & Tettamanti (1939)[25]	M	59	Abdominal pain and palpable tumor	Benign	Resection—Recovery
Mouchet & Samain (1939)[22]	F	73	Abdominal pain	Benign	Resection—Recovery
Miller & Frank (1939)[24]	F	72	Abdominal swelling and distress	Malignant	Excision — Recovery. 6 mos. after operation without complaint
Miller & Frank (1939)[24]	M	47	Loss of weight and abdominal discomfort	Malignant	Resection. Death 3 mos. later
Bonniot (1939)[26]	M	64	Abdominal pain and palpable tumor	Malignant	Resection. Death from recurrence
Colillas & Masciottra (1940)[28]	F	46	Palpable tumor	Benign	Resection—Recovery
Schousboe (1941)[29]	F	51	Symptoms of acute appendicitis	Benign	Resection—Recovery

of the pathologic report of these cases, 12 were found to be benign and six were malignant. All of these cases, with the exception of one, were of the solitary type.

There is no uniformity in the clinical details furnished by those who have reported these cases of intestinal neurofibromata. The growths had in all probability existed for a long time without producing any symptoms whatsoever. The youngest patient was 29; the oldest 73; the average age being 53.5. There were seven males and 11 females. Some of the patients gave a history of gastro-intestinal disturbances which may well have been due to the presence of the tumor, but none of these manifestations could be considered pathognomonic. In some of the cases cited there was a personal or familial history of von Recklinghausen's disease, but this did not occur often enough to establish any positive pathologic background upon which a characteristic clinical picture could be projected. Abdominal pain and palpable tumor were the most frequent complaint, the former being present in eight patients and the latter in seven. Appendicitis would appear to have been the preoperative diagnosis in two of the cases. In four cases exploration was performed on account of intestinal hemorrhages. Intestinal obstruction occurred in three cases, including

my own. In several instances peritonitis was evident when the patient came under examination, but its cause was determined only after the abdomen had been opened. Perforation of the bowel took place in several cases where the tumor had become necrotic, or had attained so great a size as to cause pressure atrophy or necrosis in some adjacent structure.

Case Report.—P. L., colored, male, age 65, was admitted to the hospital, January 8, 1939, complaining of pain in the lower abdomen of 48 hours duration. The pain was colicky in character and had grown progressively more severe. Purgatives had been given, but the patient stated he had been unable to retain them. There had been fair results from several enemata, but no relief from the pain. Twenty-four hours before admission he had begun to vomit, and had been able to retain nothing thereafter. The patient's health had been fairly good except for an occasional gastro-intestinal upset. He had, however, noticed a mass in his lower abdomen which had been present for about a year. As it was not painful he had ignored its presence.

Physical Examination.—The patient was a somewhat emaciated colored man, weighing about 135 lbs., and who appeared older than his stated age. His knees were drawn up against his greatly distended and tympanitic abdomen, and he appeared to be in intense pain. Despite distention, the mass could easily be palpated in the middle line. Roentgenologic examination, after the administration of a barium enema, revealed no abnormality in the colon or sigmoid. Except for many defective teeth, his head, chest and extremities were negative. Blood pressure 120/80; temperature 98.6° F.; pulse 90; respirations 30; R. B. C. 4,320,000; W. B. C. 12,150; hemoglobin 88 per cent; polymorphonuclears 83 per cent; lymphocytes 17 per cent. Uranalysis was negative save for a trace of albumin. Kahn and Wassermann tests were negative. *Preoperative Diagnosis:* Acute intestinal obstruction, complicating an intra-abdominal tumor of unknown character.

Operation.—A lower midline incision exposed a solid tumor, ovoid in shape, the size of a small grapefruit, attached to the periphery of the small intestine in the region of the lower ileum by a pedicle 2 cm. in length. The growth arose from the bowel wall and was entirely outside the intestine, not encroaching upon the lumen in any way. It was not adherent to any structure, but was lying entirely free in the abdominal cavity. By twisting on its pedicle, which was about 1 cm. in diameter, the tumor had so rotated the section of intestine to which it was attached as to bring about a volvulus (Fig. 1). Though the entire loop of intestine was enormously distended, no circulatory damage had occurred. The pedicle was clamped, cut and ligated, and the tumor was removed. This released the volvulus and permitted the fecal current to resume its flow. The abdomen was closed without drainage, and recovery was prompt.

Pathologic Examination.—Gross: Dr. Arnold F. Strauss. The tumor measures 12 x 7.5 x 6 cm., weighs 342 Gm., has a well preserved capsule, showing on one side a smaller node about the size of a walnut. Consistency of tissue is about that of liver, the cut surface being unstructured, of a pink and grayish-white color, one pole being of a darker pink, with a dark red margin (Fig. 2).

Microscopic: The section shows fascicles of fibers with rod-shaped or slightly oval nuclei, which tend to palisade structure (Figs. 3 and 4). Upon cross-section the protoplasm of the fibers is often slightly fibrillary or foamy. Extensive areas are edematous, with regions of hemorrhages, round cell infiltration and occasional leukocytes. Many capillaries are present, some vessels being much dilated and showing thrombosis. von Gieson's staining reveals a very few fine collagen fibers which often surround the fascicles. *Pathologic Diagnosis:* Neurofibroma of the small intestine. This diagnosis is based on the palisade structure of the nuclei, together with their oval shape, and upon the scarcity of collagen fibers, as demonstrated by Masson, Gosset, and others.

The difficulties of comparing my own case with those previously reported will be evident to anyone who has familiarized himself with the literature, which my necessarily incomplete review has shown to be highly controversial and inconclusive. There is agreement, however, upon certain main points, as, for example, the structure of the nerve trunk from which the growths under discussion take their origin. This trunk is made of *axones*—that is nerve cell processes—which are enclosed in a sheath, the *neurilemma* or sheath of Schwann. The axones sometimes (but not always) have a myelin coating as well, but are regularly surrounded by a network of extremely delicate connective tissue—the *endoneurium*. The small bundles of nerve fibers which the endoneurium supports assume an arrangement of strands or funiculi about which is another connective tissue covering—the *perineurium*. Surrounding and supporting all the structures of the nerve trunk is a connective tissue envelope or sheath—the *epineurium*. Thus, it appears possible for new growths to arise from either of two structures—the neurilemma itself or the connective tissue which surrounds it. Embryologists are in agreement as to the process by which these nerve elements develop during fetal life. The neurilemma or sheath of Schwann, as well as the nerve cell process, are of epithelial origin, while the connective tissue, the epineurium, perineurium and endoneurium are derived from the mesoderm. But from this point we find no agreement as to just how or why the growths we are now considering make themselves manifest in the nerve tissues. Masson, as we have already seen, believes that the cells of the neurilemma are the parent tissues. While this explanation is perhaps the most logical offered for any single point of origin, to take a more conservative position and admit the possibility of their growing not only from the sheath of Schwann, but also from the connective tissue, makes for a simpler and more workable hypothesis.

From the most generally recognized pathologic description, neurofibromata as they appear in the small intestine are regularly benign, though they occasionally manifest a low degree of malignancy. The mass is encapsulated and frequently pedunculated, and most of the specimens are ovoid or spherical. If much connective tissue stroma is present the growth will be hard, and, in general, it may be stated that the firmer and harder the mass the more benign is its character. Those which resemble brain tissue in color and consistency are liable to be malignant, and though nearly all growths show some cavities, the benign ones are more firm and solid than those giving evidence of malignancy upon microscopic examination.

Under the microscope the cells appear in whorls or more commonly in bundles which interlace, with a very frequent palisade arrangement which Masson, and those of his school of opinion, regard as the distinctive appearance peculiar to growths coming under the classification "neurofibroma." The cells are stellate and elongated, while in certain spots they will be plainly polygonal. Because the walls of the abundant blood vessels are extremely thin, rupture is almost regularly evidenced by extensive hemorrhagic areas, some of the cavities which are lined with endothelium appearing to be filled to overflowing

with blood, which may also have infiltrated into the surrounding connective tissue stroma. Miller and Frank[24] make the interesting observation that as motor paralysis seldom occurs as an accompaniment to these particular nerve tumors it would indicate that they arise only on sensory nerves, so "if this reasoning is correct it may explain the infrequency of neurofibromata in the gastro-intestinal tract, since its sensory nerve supply is meager." Because of its great rarity and protean clinical manifestations this particular neoplasm will no doubt continue to vex diagnosticians whenever it occurs. The question of classification has more than academic significance, as it bears largely upon the form of treatment to be adopted. Benign growths may be removed by simple resection; or, as in my own case, merely by severing the tumor's attachment to the bowel. Even those of large size which have induced pressure necrosis can be handled without wide resection or elaborate precautions against recurrences which even a suspicion of malignancy entails.

SUMMARY

1. All benign tumors of the small intestine are rare.

2. Neurofibromata of the small intestine were first reported at the end of the nineteenth century, but the existence of neurofibromata in subjects not affected with generalized neurofibromatosis was not recognized until recently.

3. Exact pathologic classification of these new growths has yet to be established.

4. The pathologic diagnosis is based upon the palisade structure of the cell nuclei, the oval shape of the cells, and the scarcity of collagen fibers.

5. Eighteen cases which have been reported since 1929 have been analyzed.

6. One additional case is reported and described.

7. The importance of differentiation between benign and malignant growths of similar structure is emphasized, because of the ease with which benign growths may be surgically treated in contrast to the difficulties and dangers inherent in handling any type of malignant neoplasia.

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MASSIVE RESECTION OF THE SMALL INTESTINE*

REPORT OF TWO CASES

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THE PURPOSE of this paper is to discuss briefly massive resection of the small intestine and to report two cases, one of which survived the resection of 260 cm. of small intestine, with 31 cm. of sigmoid colon, and two subsequent major operations for reestablishing the continuity of the large intestine, who, finally, regained an excellent state of health; and the other case who survived the resection of 354 cm. of small intestine, with 40 cm. of sigmoid colon, but pursued a generally downhill course, and died four months later of nutritional disturbances and postoperative complications.

Massive resection of the small intestine is of interest both from the immediate survival of a critically ill patient from an extensive emergency operation with, not infrequently, secondary operations for establishing intestinal continuity, and later from the standpoint of nutritional disturbances following the loss of a large section of the bowel. The term is applied to those cases in which 200 cm., or more, of small intestine have been resected. This is generally estimated at one third the total length, though there must be considerable variation with individuals, and in children it is relatively a greater proportion of the whole. The most complete review is that of Haymond,[1] in 1935, in which he analyzed a series of 257 carefully selected cases. In this series the mortality was 33 per cent, but this is considered as unduly low due to the greater tendency to report the successful cases. In some cases a portion of sigmoid colon is involved and requires resection. This adds greatly to the operative risk but has no significant bearing upon the nutritional status. In this group the mortality is highest, being 65 per cent. The two cases herewith reported are of this type. Volvulus is the most common condition necessitating such an extensive resection. Among others are strangulated hernia, mesenteric thrombosis, adhesions and abdominal injuries.

Besides the length of the intestine resected, the immediate outcome is determined by the disease condition which necessitates the resection and the general health of the patient. While there appear to be some exceptions, the nutritional status is definitely affected by the relative amount of bowel removed. Discounting the operative risk and postoperative complications, a patient may be expected to regain a normal nutritional status following the removal of 33 per cent of the small intestine. Fifty per cent is given as the

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upper limit which may be borne, with a possibly satisfactory outcome. A loss greater than this is almost invariably followed by serious nutritional disturbances of varying degree.

Inadequate compensatory changes following massive resection of the small intestine result in loss of weight, anemia and diarrhea, and at times, edema. There is excessive loss of fat and protein in the stools. The blood calcium and serum proteins are low. In a remarkable case reported by West, *et al.*,[2, 3] in which only three feet of jejunum remained, there was noted at a subsequent operation considerable dilatation and hypertrophy of the remaining bowel. Extensive metabolic studies led to the conclusions that carbohydrate is well assimilated, protein less well, and fat only poorly. The large amount of fatty acid in the stool apparently carries with it calcium in the form of calcium soap, leading to a negative balance of this mineral. Their patient did best on a high carbohydrate, adequate protein, low fat diet with additional calcium and vitamin D. Observations of others are along the same lines.[1, 4]

CASE REPORTS

Case 1.—Hosp. No. R 110430-4511: W. A., Negro, male, age 31, was admitted to the Roper Hospital May 7, 1940, at 10:30 A.M., complaining of severe pain in the abdomen, which had begun in the epigastrium at 5 A.M. There was vomiting at the onset but none subsequently. The pain was rhythmic in character and varied in severity, but was always present in some degree. His general health had been good and there was no history of indigestion or of any previous attack. He was well-developed, well-nourished, and had an ashen grey complexion. He was obviously acutely and critically ill. He was writhing with pain. Temperature 96° F., pulse 70, and B.P. 120/80. His abdomen was moderately distended and tympanitic. There was a general tenderness and rigidity. There was no visible or audible peristalsis. Rectal examination revealed some fullness in the rectovesical pouch. W.B.C. 15,500, with 91 per cent polymorphonuclear cells. A specimen of urine could not be obtained. Roentgenologic examination revealed some fluid levels in the small intestine and gaseous distention of the large bowel up to the splenic flexure. *Clinical Diagnosis.*—Intestinal obstruction, with strangulation.

Operation.—5 P.M. Spinal anesthesia with pontocaine 20 mg. was given. A few minutes after the subarachnoid injection the patient had generalized severe convulsions, respiration ceased, and the pulse became imperceptible. He reacted somewhat to artificial respiration, inhalation of oxygen and carbon dioxide, and injections of coramine and ephedrine, though he remained in shock throughout the operation. A right paramedian incision was made. All of the small intestine in view was of a dark, blue color, likewise its mesentery. The gangrenous portion was resected with its mesentery. A section of the gangrenous large bowel was twisted with the small intestine in such a manner that it had to be divided before it could be sufficiently mobilized to permit of resection. An end-to-end open anastomosis was made between the proximal end of the small intestine and the distal end, which was 6 cm. from the cecum. A Witzel enterostomy was made proximal to the anastomosis, bringing the tube out through a stab wound on the right. The appendix was removed. The distal end of the large bowel would not reach the abdominal wall so it was closed by inversion and dropped into the pelvis. The proximal end of the large bowel was brought out through a stab wound to the left of the umbilicus to act as a colostomy. The incision was closed with catgut for the peritoneum and fine alloy steel wire for the aponeurosis. The operation took one hour and forty-seven minutes.

FIG. 1.—Case 1: Appearance of patient 18 months after operation.

The patient was returned to the ward in fair condition. The specimen removed, as measured 15 hours later, consisted of 260 cm. of gangrenous small intestine, with its mesentery, and two segments of gangrenous colon, measuring 19 and 12.5 cm., respectively. The appendix had a thick serosa and a scarred wall.

Postoperatively the patient was given continuous stomach suction and lavage through a Wangensteen tube. Sodium sulfapyridine was administered intravenously. The abdomen remained flat and the convalescence was remarkably smooth. On May 10 the enterostomy began discharging freely, and on May 13 the colostomy did likewise. The enterostomy spontaneously closed following removal of the tube on May 17. On May 20 there was noted some purulent discharge and slough at the upper end of the incision. On June 19 roentgenologic examination with a barium enema revealed the distal end of the colon to be 20 cm. from the anal orifice.

On June 25, under spinal anesthesia, in preparation for anastomosing the ends of the sigmoid colon, numerous dense adhesions were severed from around the proximal end, and a double barrel transverse colostomy was made. On July 16, under spinal anesthesia, through a low left rectus incision dense adhesions were freed, and with difficulty the ends of the colon were mobilized for anastomosis. This was accomplished by attaching a stout ligature to the proximal end and telescoping it into the distal segment by a forceps passed through the anal orifice. No other method of anastomosis was possible due to the fixation and induration of the distal end, which had to be split to permit passage of the proximal end. Following this operation there were several brief periods of diarrhea, which were readily controlled by dietary regimen.

Until August 15, a period of one month, when dye instilled into the distal loop appeared at the anal orifice, there was complete obstruction at the site of anastomosis. Later a Levine tube was passed from above, and subsequently dilatation was effected by passing bougies from below. From October 1 to November 17, the colostomy spur was crushed in stages. On November 22, the colostomy was closed under local anesthesia. On November 24, four months after the anastomosis, there was a bowel movement by the rectum. On November 30 the patient was discharged from the hospital. His general condition was good. He was eating the ward diet. He was passing several poorly-formed stools a day. The abdominal incisions were firmly healed. Laboratory examinations were as follows: W.B.C. 8,075; Hb. 10.5 Gm. Urine: Albumin one plus; blood serum protein 8.20 Gm.—albumin 4.7 Gm.—globulin 3.43 Gm.—chlorides 495 mg.—blood Wassermann—positive.—Stools: Yellow and putty-like, muscle poorly digested.

On December 29, 1941, he returned to the hospital for observation at our request. He had been enjoying good health and working at his former occupation as a laborer in a fertilizer factory. He paid no particular attention to diet. He preferred carbohydrates and proteins, but ate fat without ill effect. He had no digestive disturbance or abdominal pain. His weight was 138 lbs., a very good average for him. He had one bowel movement a day. Roentgenologic examination revealed no abnormality other than a shortening of the small intestine and a correspondingly short emptying time. Examination of the urine was negative. W.B.C. was 7,700, hemoglobin 12 Gm.

Blood sugar 81 mg.—calcium 12 mg.—phosphorous 8.25 mg.—total serum proteins 8.25 Gm., albumin 4.3 Gm., and globulin 3.9 Gm. The stools were yellow and mushy.

He was last examined on October 10, 1943. He was working as a laborer for a railroad. His general health was good. He was having two or three bowel movements a day. He paid no attention to dietary restrictions. The stools were well-formed and normal microscopically. Blood calcium 10 mg.,—phosphorus 3.5 mg.,—total serum protein 7.61 Gm.,—albumin 4.88 Gm.,—globulin 2.73 Gm.; and hemoglobin 12 Gm. per 100 cc.

Case 2.—(Reported by courtesy of Dr. Frederick E. Kredel): (Hosp. No. R 8382): J. S., Negro, male, age 56, was admitted to the Roper Hospital at 11 A.M. May 16, 1942, complaining of abdominal pain. He had been awakened at 1 A.M. by a sudden, sharp pain in the region of the umbilicus. The pain became steady but varied in intensity in rhythm, each cycle lasting 15 minutes. He had vomited continually since the onset. He had no passage by rectum. He had been losing weight the past few months and had noticed an increasing tendency to constipation. He appeared acutely ill and had an anxious expression. Pulse 70, temperature 97.4° F.,—B. P. 60/40. The abdomen was considerably distended and tympanitic, no bowel pattern was visible. Rectal examination was negative. In view of a questionable pericardial friction rub, an electrocardiogram was made. It showed changes best explained by anoxemia incident to peripheral circulatory collapse. W.B.C. 15,400, with 88 per cent polymorphonuclear cells. *Clinical Diagnosis:* Intestinal obstruction from mesenteric thrombosis. For the purpose of investigating the cardiac condition and of treating the shock, operation was deferred until 10 P.M.

Operation.—Under nitrous oxide-oxygen-ether vapor anesthesia, a left lower paramedian incision was made. Two quarts of foul-smelling bloody fluid was aspirated. All but the proximal seven feet of small intestine and the terminal ileum was gangrenous, also two feet of sigmoid. The condition was apparently due to a volvulus. The gangrenous bowel was resected. A side-to-side anastomosis of the small intestine was made. The distal end was closed and a tube was inserted through the proximal end to act as an enterostomy. The ends of the large bowel were sutured side-by-side and brought out as a double barrel colostomy. Eight grams of sulfanilamide powder were sprinkled in the abdominal cavity. The wound was closed with chromic catgut and through-and-through silkworm gut sutures. The operation lasted one hour and eighteen minutes. At the end, the condition of the patient was very poor.

The specimen removed at operation consisted of 354 cm. of gangrenous small intestine, and 40 cm. of large intestine, with vascular engorgement, hemorrhage and edema.

For several days the general condition of the patient was very poor. He was disoriented, excited and resistive. His abdomen remained scaphoid. The ileostomy tube drained freely. On May 24 a crushing clamp was applied to the colostomy spur. On May 25 the enterostomy tube was removed. On June 4 it was noted that there was profuse discharge through the fistula at the ileostomy site. The general course was downhill. For the next six weeks the intestinal drainage was profuse and caused excoriation of the surrounding skin. The patient was oriented but persisted in being resistive and uncooperative. On August 6 the fistula was much smaller and the patient was discharged to the Out-patient Department. He was eating well but his state of nutrition was poor. He had four or five soft, yellow, foamy bowel movements a day. Total serum protein was 6.99 Gm.,—serum albumin 4.91 Gm.,—serum globulin 2.08 Gm.,—chlorides 539 mg.,—urea nitrogen 16 mg.,—blood Wassermann positive. W.B.C. 7,600, polymorphonuclear cells 66 per cent. Hb. 8.5 Gm. Urine: Albumin three plus, W.B.C. 15, R.B.C. 3 per h.p.f.

On September 4, 1942, he was again admitted to the hospital with a diagnosis of intestinal obstruction. He had noticed increasing abdominal distention during the past week, though his bowels continued to move and the intestinal fistula continued to drain.

He was irritable, emaciated and anemic, but in no acute distress. The abdomen was drum-tight. He did not improve under treatment by continuous stomach suction. The blood chemistry findings were the same as on discharge August 6. On September 11, under spinal anesthesia, operation was performed for the purpose of relieving the obstruction and closing the fistula. A right rectus incision was made in line with the fistula. About five liters of ascitic fluid was found. The intestines were edematous but not discolored or much distended. The terminal ileum was kinked by dense adhesions. The fistulous tract was complicated involving the colostomy and terminal ileum. After considerable dissection the openings in the intestines were closed. On September 12 distention reappeared. The patient developed peritonitis, and died September 15, 1942.

COMMENT.—Case 1 is of interest from the standpoint of the patient's regaining good health following the resection of approximately 40 per cent of the small intestine, with a section of sigmoid colon, and two subsequent major operations for reestablishing intestinal continuity. No hernia resulted, though three longitudinal incisions were made in a contaminated field. This is partly attributed to the use of alloy steel wire. The telescoping anastomosis of the rectosigmoid resulted in stricture formation, which was dilated only with difficulty but showed no tendency to recur. Examination three years later indicated that he had regained a state of normal health.

In Case 2 the patient survived removal of approximately 53 per cent of the small intestinal and a section of large bowel. Though he lived for four months his course was generally downhill. His nutritional disturbance was increased by a small intestinal fistula which drained more or less profusely for six weeks after operation, but it did not improve following diminution of the drainage. Edema appeared early. That the blood serum proteins were not lower is probably accounted for by the state of dehydration. The nutritional changes were those to be expected. The development of partial intestinal obstruction necessitated operation four months later. Of particular interest was the finding of a large amount of ascitic fluid and edema of the intestines.

SUMMARY

1. The subject of massive resection of the small intestine has been reviewed.

2. Two cases are reported, one regained a normal state of health, the other died of nutritional disturbances and postoperative complications after four months.

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SIMPLE PENETRATING ULCER OF THE CECUM*

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SIMPLE ULCER OF THE CECUM, a lesion not associated with other recognized infections nor tumefactions of the large bowel, is usually found near the ileocecal valve on the mesial side of the cecum and has a marked tendency to perforate and to cause extensive scarring and contraction. While so-called "simple" ulcers of the colon were first described by Cruveilhier a century ago, the condition is still rarely reported, still presents a clinical picture so confusing that no case has ever been clearly differentiated from other lesions of the ileocecal coil before celiotomy or autopsy, and is still completely obscure as to its etiology.

The first collected report of nonspecific colonic ulcers was made in 1902 by Quenu and Duval, who reviewed 27 cases, including in their series lesions from all portions of the large bowel. Barron's collection of 53 cases, presented in 1928, also attempted the correlation of ulcers from this comprehensive field. In each of these reports, cecal lesions far outnumbered those in other portions of the bowel; moreover, the cecal cases, when analyzed by the reader, suggest a definite clinical entity more clearly than the ulcers found in the left colon, since we now understand that ulcerative colitis, amebiasis, diverticulitis and other disease processes partial to the left colon could have been responsible for the lesions found there.

It is perhaps for this reason that recent surgical literature reveals a distinct tendency to segregate the single, nonspecific, penetrating ulcer of the right colon as more nearly answering the essential criteria for a pathologic entity.

Males of middle age are more prone to this condition, although it has been encountered in both sexes and in individuals from 17 to 80 years old. The symptoms of the disease are so variable and pathognomonic signs so lacking that continued difficulty in specific diagnosis is understandable, but it is fortunate that the clinical manifestations which are present clearly demand surgical intervention, as no medically treated individual has survived.

In the acute form, where perforation has occurred or is imminent, the symptoms are apparently indistinguishable from those of appendicitis, in fact, Barlow states that in 68 per cent of those cases in which diagnosis has been attempted, the abdomen has been explored under that misconception. Dickenson, who encountered at long intervals three individuals with cecal ulcer, stated that he operated upon each in turn with the preoperative diagnosis of appendicitis. Excision and suture of the ulcer, or suture alone has been used successfully by Cameron, Barlow, Harrison, and others, when

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the ulcer evidenced recent perforation without tumefaction of the bowel, but when drainage alone was relied upon the outcome has invariably been fatal.

When the perforative process is slow, the associated fibroplastic reaction gradually produces extensive tumefaction of the cecum, ascending colon and even the nearby ileum, and when the omentum attaches itself to the ulcer bed a similarity to neoplasm becomes even more marked. In this chronic latent stage constipation is a common symptom, vague abdominal pain, low-grade fever, slight tenderness over the mass in the right lower quadrant and, more rarely, intestinal bleeding may be encountered. Since the ileocecal coil is the location of the great majority of those inflammatory conditions of the abdomen which require surgical treatment, as well as the site of one-fourth of colonic cancers, it is evident that a variety of pathologic lesions can present signs and symptoms similar to those of chronic simple ulcer of the cecum.

Periappendicitis may of course give rise to the firm, woody pseudotumor described by Walters and Synhorst as "ligneous" infection of the cecum. Hypertrophic tuberculosis of the cecum, frequently a primary lesion and causing mild local tenderness, discomfort, indigestion and occasional bleeding from the bowel, not only produce a similar mass but presents the same roentgenologic picture of constriction, as the process is associated with excessive fibrosis and consequent thickening of the bowel wall. The chronic granulomatous disease, regional ileitis, begins by preference at the ileocecal valve and extends upward along the terminal ileum, but direct extension to the cecum may occur. In the chronic form a palpable mass is present, consisting of soggy, edematous, adherent bowel and omentum. Diarrhea, fever, extreme chronicity and a tendency to occur in younger individuals should, of course, differentiate this lesion from ulcer. Cecal cancer is frequently palpable although, in theory, the absence of pain and fever and the presence of anemia should aid in its differentiation. The roentgenologic examination is not always helpful, however, as a malignant cecal tumor not infrequently produces a constricting defect.

A review of the chronic or latent cases reported discloses that cancer has been the most common preoperative or postoperative diagnosis and that resection of the right colon has usually been considered advisable.

My own observation of this disease includes two cases, both seen in the latent phase:

CASE REPORTS

Case 1.—C. H. H., white, male, age 68, was transferred to the Proctologic Service of the City-County Hospital, February 2, 1941, with the chief complaint of indefinite pain in the right lower quadrant for 18 months. The discomfort had been much more severe for three months, and periodic attacks associated with abdominal cramps during that time had been relieved by vomiting. No blood had been seen in the stool, and there had been little temperature elevation, but the patient had observed an increase in constipation.

Examination disclosed a firm, movable, palpable tumor in the right lower quadrant,

with no rigidity, no fever, slight leukocytosis, and no anemia. A colon roentgenogram was done (Fig. 1), and the following observations made: "Marked deformity of the cecum, chiefly narrowing near the ileocecal valve; movable tumor in this area; spasm apparently associated with the tumor. *Impression:* Organic constrictive lesion of the cecum. Neoplasm considered first, hyperplastic tuberculosis second, and specific inflammatory process third."

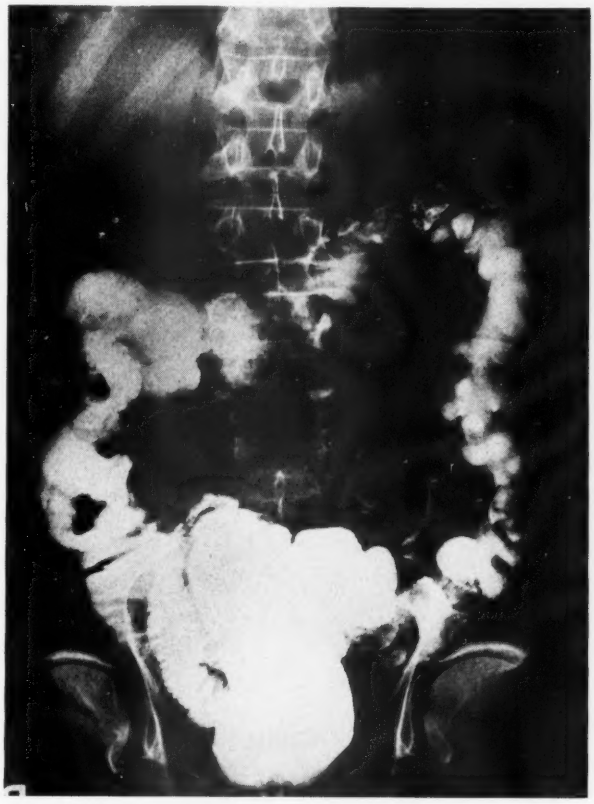

FIG. 1.—Case 1: Radiograph showing spasm and constriction of ascending colon and cecum.

On February 12, 1941, the abdomen was explored, with the preoperative diagnosis "cancer of the cecum." A sausage-shaped tumor was found; no change in the liver nor abdominal nodes was noted. An ileocolostomy was done. A rise in temperature occurred after this procedure, but subsided. On February 26, with the diagnosis "tumor" of the cecum, the right colon was resected. Grossly, a dimpled area was noted near the ileocecal valve, together with marked constriction of the cecum and a portion of the ascending colon. The tissue report was as follows: "The cecal mass measured 11 x 7 x 6 cm. In one area a definite constriction was seen; the mucosa was ulcerated and the bowel wall thickened. A portion of the wall on the medial side was deeply necrotic. Microscopic findings: Ulceration of the mucosa which was infiltrated with lymphoid and plasma cells. In the center the ulceration was deeper, at the periphery, marked fibrosis was observed. No neoplastic nor specific changes were seen. *Impression:* Chronic, nonmalignant, nonspecific solitary ulcer of the cecum."

The patient died ten days following his second operation from peritonitis induced by leakage of the intestinal suture line. An autopsy disclosed that, other than the peritonitis, no lesion of the intestinal tract nor abdomen was present.

The condition was encountered by us again in December, 1942; the patient, again, being an elderly individual with a subacute hyperplastic lesion, which had apparently partially perforated three weeks before entry into the hospital.

Case 2.—S. W., white, female, age 65, married, was first examined on December, 1942. She stated that she had noted for some months mild indigestion with flatulence, but that for three weeks a cramping pain had been present in the abdomen, especially in the right lower quadrant, associated with slight nausea. The bowel habit was

FIG. 2.—Case 2: Radiograph showing constriction of cecum. Efforts to fill this portion of the bowel with barium were unsuccessful.

unchanged, there was an increase in the leukocyte count but the red cell count was normal. A mass was palpated in the right side. A roentgenogram demonstrated a filling defect in the cecum, associated with constriction (Fig. 2).

At exploration, under the preoperative diagnosis "cancer of the cecum," this portion of the bowel was found enlarged, firm and surrounded by omental adhesions. An ileocolostomy was done, which was followed by a stormy convalescence and irregular temperature.

Several weeks later, under the rather evasive preoperative diagnosis "tumor of the cecum," the abdomen was entered again. Some free fluid was found but conditions were otherwise unchanged. The right colon was resected at this time. The pathologic report was as follows: "In the cecum, 2.5 cm. from the base of the appendix, there is a firm ulcerated area. The wall of the cecum is thickened and the overlying peritoneum

of the terminal ileum is thick and roughened. Microscopically, it is shown that the ulcer extends through all the layers of the bowel, its base consisting of granular tissue infiltrated with leukocytes; in the fibrotic tissue surrounding the ulcer an irregular scattered round cell infiltration is noted. Sections of the ileum reveal normal mucosa with infiltration of the serosa and submucosa with round cells; a nearby lymph node revealed hyperplasia of the reticulo-endothelial elements. No evidence of any specific etiologic change was observed in the tissue examined. *Diagnosis:* "Simple ulcer of the cecum with partial perforation and subacute pericecitis and periappendicitis."

While less than 50 cases of simple ulcer have found their way to the literature, it is probable that numerous unreported or unrecognized examples of this pathologic entity have occurred. Osler, for example, briefly and without detail, mentioned that "two instances of ulcer of the cecum, both with perforation, have come under my observation," and Whipple recently listed cecal ulcer as the cause of two resections of the right colon. While it is true that no proven etiologic agent can be assigned to this condition, this is no less true in the case of a similar phenomenon, peptic ulcer.

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DISCUSSION.—DR. J. SHELTON HORSLEY, Richmond, Va.: Doctor Collins has gone very interestingly into the subject of neurofibromas. As he said, there has been much discussion of neurofibromas of the intestinal tract, and doubtless many tumors that have heretofore been classified as myomas or fibromas or leiomyosarcomas may be found on reexamination to be neurofibromas. The palisade effect in the microscopic examination to which Doctor Collins called attention is a rather important point in diagnosis and tends to confirm the diagnosis of a neurogenic tumor.

Occasionally there are degenerating neurofibromas. A case of mine was rather puzzling. Mrs. K. W., age 39, had suffered from intestinal hemorrhages at repeated intervals for several years. Her hemoglobin was 32 per cent; R. B. C. 2,880,000. Gastro-intestinal roentgenologic examination was negative. The physical examination was essentially negative. A tentative diagnosis of duodenal ulcer was made and, on operation, there was found at the hepatic flexure of the transverse colon, a flat mass arising from the transverse mesocolon and attached to the jejunum. It was extremely vascular. Part of the jejunum and a large segment of the transverse colon were resected. The patient made a satisfactory recovery. The tumor was a vascular, degenerating neurofibroma springing from the transverse mesocolon and invading the upper jejunum at the point which was the source of the hemorrhage (slide). The next slide

shows the microscopic appearance (x150). According to Broders, this is a degenerating neurofibroma, and is essentially benign.

Another slide is from a tumor of the stomach that I formerly classified as low grade leiomyosarcoma. The palisading effect is well shown, however. It seems probably that this tumor is a type of neurofibroma. After resection of the stomach there has been no recurrence.

DR. WALLACE FRANK, Louisville, Ky.: In 1939, Miller and I reported two cases of nerve tumors of intestinal origin before the American Association for the Study of Neoplastic Diseases. We submitted sections which greatly resembled in most detail the photomicrographs shown by Doctor Collins. It was their opinion that the cases which we reported were not simple neurofibromas but rather neurofibrosarcomas. One case we reported was truly von Recklinghausen's disease of the small intestine. There were thousands of lesions ranging in size from a pinpoint to a baseball. This man died of intestinal obstruction. At autopsy no evidence of any metastasis was found.

I would like, with Doctor Rosser's permission, to add to the literature another case of simple ulcer of the cecum. The best article I have seen on this subject was by Cameron, published in the British Journal of Surgery, January, 1939. Cameron emphasizes the fact that the ulcer is always opposite the ileocecal valve. He compares simple cecal ulcer to duodenal ulcer and states that the acidity of the material being poured through the valve may be a causative factor.

Case Report.—C. T., a man, age 23, was first seen in May, 1937. He gave a history of pain in the abdomen, nausea but no vomiting. The symptoms began 40 hours prior to the time I saw him. His temperature was slightly elevated, pulse about 96. There was definite tenderness and rigidity of the lower right rectus muscle. The white blood count was approximately 16,000, with 79 per cent polys. He was seen in the afternoon and operated upon as an emergency. At operation, the appendix was definitely reddened and congested, and was removed. It did not seem to be acute enough to account for all his symptoms; consequently I carefully explored the terminal ileum but found no pathology. Palpation of the cecum revealed a hard indurated area on its postero-external wall. The peritoneum to the outer side of the cecum was incised and the cecum turned inward. This exposed a grayish-white area about 1.5 inches in diameter and 0.5 inch thick; in the center a definite crater could be palpated. As the lesion was extraperitoneal I deemed it unwise to excise it. There was not enough peritoneum over the cecum to do a resection of the cecum, and as the patient had not been prepared for any radical surgery I thought it inadvisable to do a resection of the right colon. The cecum was replaced and the peritoneum sutured.

Following operation the stools were repeatedly studied for amebae, Bargen's bacillus and tubercle bacillus; none were found. I emphasize the fact that we looked especially for amebae because we had seen marked cecal deformity due to amebiasis.

This patient has been under observation for the past six years. Occasionally he has discomfort in the right lower abdomen and on two occasions there has been intestinal bleeding. He feels well, has had no medical treatment and has refused further surgery. I would like to show the roentgenogram of the cecum. The original films taken in 1937 have, unfortunately, been destroyed. This film was made six weeks after he left the hospital but is similar in character to the one taken ten days after operation.

DR. ROGER G. DOUGHTY, Columbia, S. C.: In listening to Doctor Rosser's paper I was reminded of some cases which occurred in my work some years ago. I must apologize for not having accurate data to present. Doctor Rosser tells me these cases are extremely rare.

One patient in particular, about 60 years of age, had a long history of difficulty with the intestinal tract. He had been accused of amebic dysentery but it had never been demonstrated. He had also been under medical treatment for various intestinal upsets, labeled with various names. When I saw him he had what I took to be an acute appendicitis. He was sent to the hospital and, with the usual preparation, was operated upon. I was under rather unusual stress because this was the uncle of my wife, a doctor, his son was a doctor and was present. I was quite confident that I was dealing with carcinoma. We did a resection and end-to-end anastomosis of the ileum

to the stump of the large bowel. The head of the cecum was covered with a small mass of fibrin about 1.5 x 2 cm. long. The wall was very much indurated, the area of induration extending over about 6 cm. from the base upward, almost completely around the bowel, and the fat tips were indurated.

The report from the pathologist indicated nonspecific ulcer. We sent the specimen to another pathologist who came back with essentially the same report. Because of there having been the suspicion of amebic dysentery we checked the pathology again, and found no evidence that would make either pathologist think it was really an amebic lesion.

Just about three weeks ago another patient came with what I thought was acute appendicitis. Again we opened the abdomen and found a normal appendix. About opposite to the ileocecal valve was a moderately thickened area in the cecum. When we wiped the cecal wall with gauze it stippled. We took the appendix out, closed the wound and the patient was sent back to bed; she was relieved of her symptoms. These things must have a beginning, and Doctor Rosser's paper makes me wonder if I stumbled on the beginning of a benign ulcer.

Dr. R. L. Sanders, Memphis, Tenn.: Doctor Prioleau has raised an interesting point in reporting his two cases of massive resection of the small bowel. He has shown that a large portion can be removed without altering its function or materially endangering life. This has likewise been true in our experience. I recall one patient, a woman, age 74, who came to us for acute mesenteric thrombosis. We removed more than six feet of the small bowel. She recovered completely and has had no functional difficulty since the operation, now eight years ago.

The essayist also stated that one of his patients developed a troublesome fistula following a complementary enterostomy proximal to the anastomosis. Formerly, we had one or two cases in which a similar complication developed, and a few others wherein the bowel became adherent at the site of the enterostomy, resulting in intestinal obstruction. During the past several years, however, we have obviated these possibilities by omitting enterostomy in all our resections of both the small intestine and the right half of the colon. After reestablishing the continuity of the bowel by an end-to-end or side-to-side anastomosis, we have done nothing more. The use of a simple indwelling tube, as advocated by Wangensteen, has sufficed to prevent postoperative distention and consequent tension on the suture line.

Dr. J. M. T. Finney, Baltimore, Md.: These so-called massive resections of the small intestine may at times be multiple. I have a patient upon whom I operated about three months ago, who, seven years ago had about five feet of terminal ileum resected by another surgeon because of a mesenteric thrombosis. About three months ago I was called in and resected three feet eight inches of the remaining ileum because of gangrene due to volvulus caused by adhesions from the previous operation. This makes a total resection of approximately nine feet in two sittings. She is perfectly all right, and much to my surprise does not have diarrhea.

SURGICAL INDICATIONS IN DISEASES OF THE COMMON BILE DUCT*

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SURGERY of the bile ducts has, within recent years, assumed a new aspect. Much of the progress in this field has been due to a better understanding of surgical principles and the indications for exploration. Further, a more accurate determination of the pathologic condition within the ducts has been made possible by cholangiography, and many of the hazards of operation have been reduced by the use of vitamin K and the sulfa drugs. Despite these facts, the mortality of surgical procedures on the ducts remains high, largely because of the almost universal association of disease of these structures with advanced cholecystitis and liver damage. We may look forward to the time when an operation upon the ducts will be a comparatively rare incident because of early surgical termination of gallbladder lesions, but until then we can only continue to apply the principles of good surgery to the individual case and make the best possible use of the therapeutic adjuncts at our command.

The chief conditions for which operations on the ducts are performed are: 1. Stones. 2. Carcinoma. 3. Stricture. 4. Pancreatic lesions.

In this discussion, no attempt will be made to describe in detail the clinical picture of each of the foregoing pathologic conditions. Instead, our own experience with duct disease during the past eleven years will be presented, including our conception of the indications for choledochotomy, our methods of management, and the results obtained.

During these eleven years, we have performed 596 operations upon the biliary tree, 575 of this number having been for benign disease of the gallbladder and ducts. Of the 575, 538 were primary operations and 37 were secondary. The ducts were opened and explored 77 times, or 13.4 per cent of the 575. Sixty-one of the choledochotomies were primary and 16 were secondary. There were 17 fatalities, or 3.4 per cent following the gallbladder operations without exploration of the ducts. Compared to this, there were eight fatalities, or 10 per cent, following cholecystectomy or cholecystostomy with choledochotomy. Thus, the mortality rate was almost three times higher in the latter group. These findings are shown in Table I.

Of the 575 operations, stones were found in the gallbladder in 328, or 57 per cent, and of the 77 choledochotomies, duct stones were found in 54, or 70 per cent, as will be seen in Table II.

The mortality rate from the choledochotomies wherein stones were found was 6.8 per cent as compared to 2.8 per cent for those in which no stones

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TABLE I

OPERATIONS IN 575 CASES OF GALLBLADDER AND DUCT DISEASE

		Mortality
Primary gallbladder operations without choledochotomy	477	15 (3.1%)
Secondary gallbladder operations	21	2 (9.5%)
Total	498	17 (3.4%)
Primary gallbladder operations with choledochotomy	61	4 (6.5%)
Secondary choledochotomy	16	4 (25%)
Total	77	8 (10%)

TABLE II

INCIDENCE OF DUCT STONES IN 575 OPERATIONS FOR GALLBLADDER AND DUCT DISEASE

Gallbladder operations		575
Stones	328 (57%)	
Duct operations		77 (13.4% of 575)
Stones	54 (70% of 77)	

were recovered. We do not believe, however, that the presence of stones has any bearing upon the outcome except insofar as they are usually associated with long standing disease.

In an attempt to compare the number of our choledochotomies and findings of duct stones with those of other surgeons, we have compiled a few figures from reported series (Table III).

TABLE III

	Total Opers.	Choledochotomy	Duct Stones
Allen and Wallace (4 years)	266	159 (59.7%)	61.6%
Cutler and Zollinger (5 years)	397	145 (36.5%)	51%
Glenn (6 years)	907	100 (11%)	55%
Heyd (13 years)	3,306	260 (7.7%)	86%
Lahey Clinic (32 years)	3,373	1,292 (38.3%)	37%
Mayo Clinic (1 year)	812	182 (22.3%)	60%
Sanders (11 years)	575	77 (13.4%)	70%

The series reported by Cutler and Zollinger, Glenn, and Heyd were all hospital cases, whereas the remainder were private clinic cases. In this connection, Cattell reports that in the Lahey Clinic during the four-year period of 1938 to 1941, the common duct was explored in 444 of 909 gallbladder cases, or 48.8 per cent, and stones were recovered in 128, or 29 per cent of the explored group, and 14.1 per cent of the 909. Further, during the year 1942, they explored the duct in 130, or 45.3 per cent, of 287 gallbladder cases, but found stone in only 28, or 21 per cent of the 130 choledochotomies, and 9.8 per cent of the entire group of 287 cases.

Until a few years ago, our criteria for openly exploring the ducts in cholecystic disease consisted largely of the palpation of a stone and jaundice. Lately, however, we have been guided by the following findings: 1. Palpation of a stone in the duct. 2. Jaundice, or a history of jaundice. 3. A contracted gallbladder. 4. Abnormal dilatation of the ducts. 5. Aspiration of flocculent bile from the duct.

For purposes of comparison, and to show our more recent experience, we have divided the total of 538 primary gallbladder operations as shown in Table I (477 without choledochotomy and 61 with choledochotomy), and the 37 secondary operations (21 on the gallbladder and 16 on the ducts) into two groups: Those performed prior to June, 1941, being 383 in number, and those performed since that date, numbering 192.

TABLE IV

COMPARATIVE REVIEW OF 575 GALLBLADDER AND DUCT OPERATIONS

	Gallbladder Operations	Gallstones	Duct Operations	Duct Stones
Group I.....: 10/1/32–6/1/41	383	215 (56%)	35 (9% of 383)	26 (74% of 35)
Group II.....: 6/1/41–10/1/43	192	113 (59%)	42 (21% of 192)	28 (66% of 42)
Total.....:	575	328	77 (14.3%)	54 (70% of 77)

Our mortality in the first group was 5 per cent; in the second group, 3.1 per cent.

As will be observed, in the first group our duct operations were considerably fewer in proportion than in the second group, being 35, or 9 per cent of the 383 gallbladder operations performed over a period of almost nine years. In the second group of 192 operations within a little more than two years, we have openly explored the duct in association with operations for cholecystitis 42 times, or 21 per cent. In the first group, stones were found in 74 per cent of the 35 choledochotomies, whereas they were found in 66 per cent of the second group of 42. Although the percentage of choledochotomies was much higher in the second group, the percentage of stones was less. The 28 duct stones in Group II represents approximately 15 per cent of the 192 cases of cholecystitis and 25 per cent of the 113 in which stones were found.

From this experience, we do not see the necessity of subjecting patients to choledochotomy, even though the risk is small, without clear indications. Some surgeons regard dilatation of the ducts as sufficient indication for exploration. In all our operations for cholecystitis we have routinely examined the ducts visually as well as by palpation, with the exception of a few cases wherein the process was too acute and edema too extensive to permit, and have found the common or hepatic duct or both dilated in almost 80 per cent. In 150 of the 192 cases in Group II, some degree of duct enlargement was observed, though in only 113 were gallstones recovered. The ducts, therefore, were enlarged in many cases wherein stones were not even present in the gallbladder.

Obviously, considerable discrimination must be used in performing choledochotomy when a dilated duct is found. The palpation of a stone and jaundice have been our primary indications. As is well known, however, jaundice is an unreliable sign of stone. Past or present jaundice was a finding in only 38, or 50 per cent, of our cases in which duct stones were recovered. Jaundice,

or a history of jaundice, was a factor in 86 per cent of the remaining choledochotomies, and in approximately half of these the duct was dilated because of an associated pancreatitis, usually with a more or less extensive hepatitis. Occasionally, also, we have opened a dilated duct in the presence of jaundice, only to find no explanation for the obstruction other than a possible spasm of the sphincter of Oddi.

In the absence of jaundice and a palpable stone, our criteria for opening dilated ducts have been the discovery of a contracted gallbladder; small stones in the gallbladder with an enlarged patent cystic duct; the presence of flocculent bile in the ducts as determined by aspiration; and a history of chills and fever. Not infrequently, however, the common duct is abnormally dilated incident to occlusion of the cystic duct with a stone in long-standing gallbladder disease, the duct having taken over the function of the gallbladder. The bile being clear, there is no point in choledochotomy. We have also observed a few cases in which the duct was dilated from obstruction by an enlarged node, an anomalous artery, or by adhesions. In these, removal of the obstruction has been sufficient to relieve the condition.

Some degree of hepatitis or pancreatitis, unassociated with jaundice, has been encountered in a high percentage of the cases of this entire series. As a rule, we do not open the duct in such cases, believing that the inflammatory process will subside following cholecystectomy alone. Our results have borne out the wisdom of this view.

Duct stones were recovered in ten of the 16 secondary operations on the ducts. The obstruction was caused by adhesions in one case, by stricture in three, by pancreatitis in one, and in one case apparently by spasm of the sphincter.

Of the 16 secondary operations, the primary procedure was carried out elsewhere in 12 cases and by us in four. One of the latter was in the second group of Table IV. In this case, at the first operation numerous bile cysts were found in the liver and the common duct was as large as the duodenum and filled with dark, sandy bile. The distal end of the duct was dilated to the size of a No. 7 dilator and drainage instituted. Nevertheless, stones were subsequently formed in the liver in such quantities as to pack the ampulla, necessitating a second choledochotomy and anastomosis of the duct to the duodenum in order to promote complete emptying of the bile passages.

Within recent years we have made a practice of dilating the lower end of the duct to almost normal size with Bakês dilators, when possible. No untoward postoperative effects have been observed. Combined with thorough exploration of the ducts, lavage and the use of the T-tube, and medicinal measures to stimulate bile drainage, this procedure has been ample to insure the patient against further trouble, with the above one exception. We have not considered it necessary to make cholangiograms at the time of operation, though we do so before withdrawing the T-tube when such a procedure seems indicated.

In this connection, I wish to call attention to an incident which has happened

more than once in our experience, and which has a possible bearing upon secondary operations for stones in the duct. In one case, the common duct was opened and explored, stones were removed, and the duct irrigated with salt solution before the cholecystectomy was begun. While the gallbladder was being manipulated, small stones slipped through the cystic duct into the common duct under sight, necessitating a second irrigation of the duct. In another case, the common duct was opened and evacuated of stones, and the T-tube sutured in place. The gallbladder, which contained a large number of small stones, was then removed. The cystic duct was patent and fairly large. The tube did not drain well, and it was necessary to reopen the duct and withdraw the tube. It was then found that three or four stones had passed through the cystic into the common duct. A subsequent operation might have led us to believe that the stones had reformed in the duct. Since these experiences, we have removed the gallbladder before doing the choledochotomy; thus, we can be assured that the common duct is entirely free of stones at the conclusion of the operation. If, later, it should be necessary to open the duct because of stones, we can be certain they have been formed subsequently, and are not stones which were overlooked at the first procedure.

Three patients of this series, or 0.5 per cent, came to operation for partial stricture of the duct, all following surgery. Two of the three had been operated upon by us. In one of these cases, the stricture was induced by clamping of the duct in an attempt to control hemorrhage from a necrotic, ruptured cystic artery which paralleled the duct. A choledochotomy was done and the distal end of the duct dilated. The patient has since remained well.

The partial stricture in the second case was the result of a narrowing of the duct at the site of a former choledochotomy. Three years had intervened since the operation and the liver had become involved in an advanced cirrhosis. The duct was widely explored and drained. The patient succumbed two weeks later from liver and kidney dysfunction.

The third patient had had a partial cholecystectomy elsewhere. At the second operation, the cystic duct had become enlarged, forming a miniature gallbladder, and was filled with stones, the distal end of the common duct was strictured, and a severe pancreatitis was present. The gallbladder was removed, the duct opened and explored, the distal end dilated and drainage instituted. The patient is still free from symptoms, now more than a year since the operation.

By far the majority of duct strictures arise from operative trauma incident to clamping of the ducts in an effort to control hemorrhage from an accidentally injured artery. Such accidents are especially likely when the ducts and arteries are abnormally situated. We have encountered anomalies of these structures in fully 50 per cent of our cases. One can thus readily appreciate the necessity for adequate exposure and meticulous care in their manipulation. The second of the above cases, though not itself the result of actual trauma, presents an illustration of the possible consequences of even partial stricture.

The common duct is one of the rarest of locations for malignant disease. In Heyd's series of 3,986 biliary tract operations, duct carcinoma was encountered only five times. Two, or 0.3 per cent, of the patients in the present series of 596 had carcinoma of the common duct. One, a man, age 68, gave a history of gallbladder symptoms for 30 years, and had recently developed a deep jaundice, but without chills and fever and without pain. The gallbladder was distinctly palpable. The preoperative diagnosis was carcinoma of the pancreas or duct, with possible extension to the liver. At operation, the tumor was found in the common duct at its junction with the cystic, and extending backward into the liver. The gallbladder was enormously distended with dark bile but was not diseased, and since the hepatic duct was apparently patent, a cholecystogastrostomy was performed. The patient lived but a short while after leaving the hospital.

The second patient, a woman, age 71, also gave a history of long-standing gallbladder disease and recently abdominal distress and increasing jaundice, but without chills. The gallbladder, however, was not palpable. The preoperative diagnosis was cholecystitis with stones and probably stones in the duct. On opening the abdomen, there was an extensive carcinoma in the midportion of the duct. A part of the growth was encroaching upon the gallbladder and liver. The gallbladder had perforated into the liver, evidently a long while previously, and a number of stones were contained in the pocket. As much of the growth as possible was excised, an hepaticoduodenostomy performed, and the gallbladder drained. This patient, also, died at home after a few weeks.

It will be observed that the location of the carcinoma in both the above cases precluded the use of the Whipple operation. We have, therefore, had no occasion to employ this procedure.

In the first case, the painless, rapidly progressive jaundice and the palpable gallbladder were typical of carcinoma. In the second, the diagnosis was obscured by the presence of gallstones. No duct stones were found in either case. These experiences tend to bear out the observation of other surgeons that, apparently, there is no direct association of duct stones to carcinoma of the ducts such as is observed between gallstones and carcinoma of the gallbladder.

On the whole, the prognosis for carcinoma of the ducts should be better than for carcinoma of the gallbladder or the intestinal tract, in that patients come to operation early on account of obstruction. This was not true of either of the above cases, however, wherein the growths were extensive despite the fact that definite evidence of obstruction had been present only a few weeks.

We have had two occasions to do a choledochoduodenostomy for carcinoma of the pancreas, the gallbladder having been previously removed in both cases. In an additional 12, cholecystogastrostomy was carried out. There were no hospital deaths in any of the group of 14.

COMMENT

With few exceptions, the object of operations upon the ducts is release of obstruction by a stone, and in our opinion one is not justified in adding choledochotomy to the already severe handicap of these patients without ample evidence of stones. It is agreed that when a stone is palpable, the patient is jaundiced or has a history of jaundice, or chills and fever, and the common duct is abnormally enlarged and thickened; when there are stones in the gallbladder and the cystic duct is dilated; or flocculent bile is present in the duct, pointing to a possible impalpable stone in the ampulla, then choledochotomy is definitely indicated. Mere dilatation of the common duct, however, is not in itself an indication. In many cases, the dilatation is physiologic, being due to a dysfunctioning gallbladder. In others, it is due to extrinsic mechanical obstruction. In still others, it is secondary to a gallbladder infection and is amenable to relief by cholecystectomy alone. Nor do we share the view that hepatitis and pancreatitis are, generally speaking, indications for exploration of the duct. As a rule, these conditions will subside following removal of the gallbladder.

It appears that the finding of duct stones varies in the experience of different surgeons, and from time to time in the experience of the same surgeons. In our own experience, the age incidence of patients who have come to surgery for cholecystitis within recent years has been materially lower. The incidence of stones in the gallbladder has consequently been less as patients are operated upon at an earlier age. We are, however, operating upon a larger number of patients for cholecystitis with stones and a smaller number for noncalculous cholecystitis. Thus, our percentage of gallstones remains practically unchanged. One would naturally expect that, in younger patients who have had stones in the gallbladder for a shorter period, the incidence of duct stones would be reduced and there would be a corresponding reduction in the number of choledochotomies. We believe this explains the lower incidence of duct stones in proportion to the number of explorations in our cases within recent years. Should the practice of operating upon patients earlier continue, as it no doubt will, it should lead, not to more, but to fewer choledochotomies in the future.

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CHOLESTEROLOSIS: ITS SIGNIFICANCE IN THE BADLY DAMAGED GALLBLADDER*

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THE CLASSICAL FORM of cholesterolosis of the gallbladder generally manifests itself by a submucosal deposit of esters of cholesterol chiefly in histiocytes. These deposits are often focal in character producing the gross appearance of a small yellow elevation when the mucosa is inspected in the fresh specimen. The histiocytes become large and foamy and are associated with a club-like enlargement of the villi. While these histiocytes represent true xanthoma cells, this phase of cholesterolosis is but rarely associated with giant cell reaction and fibroplasia as is often the case with focal xanthomatous reactions in other organs. The so-called "strawberry gallbladder" is commonly found associated with cholesterol stones and as such is of great clinical importance. Cholesterolosis, itself, however, has less surgical significance chiefly because of the lack of association with any great degree of inflammation in the wall of the gallbladder and thus its failure to produce severe symptoms.[1]

Because of its unique clinical and pathologic picture, cholesterolosis has been placed in a separate category in the classification of lesions of the gallbladder. Whereas, in times past there have been many who have considered cholecystitis primarily an infectious disease, there have been few who would deny that cholesterolosis is truly a metabolic disease—a chemical cholecystitis.

Where gallstones have been present for some time it is not unusual to find a gallbladder showing evidence of cholesterolosis and at the same time the more common type of fibrous cholecystitis. We are thus often confronted with a picture of cholecystitis when the two types of inflammation have definitely merged into one, that makes such a dual concept of pathogenesis awkward.

In several recent publications[2] we saw reason to doubt the frequency of primary bacterial cholecystitis and presented experimental evidence to show that other forms of cholecystitis than that of cholesterolosis or strawberry gallbladder may also be primarily chemical. Where an infectious process is present we have suggested that this exists as a secondary invasion of previously damaged tissue rather than as a primary bacterial infection. We called attention to the fact that bile itself, is damaging to tissue. It sets up an inflammatory process of a type similar to that seen in cholecystitis. It produces a marked increase in capillary permeability in the region in which it was placed. This is first evidenced by edema but later there may be even diapedesis of red blood cells and ecchymosis. Following the alteration in capillary permeability one sees definite local tissue damage in the form of actual gangrene or a lesser

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damaging effect. This inflammatory process is associated with a cellular reaction which is chiefly lymphocytic and histiocytic although a few polymorphonuclear leukocytes may be present. The severity of this inflammatory reaction produced by bile seemed to be in direct proportion to the concentration of cholesterol and bile salts.

Because of the similarity between such local effects of bile in tissue and the pathologic picture seen in the human in acute cholecystitis, we suggested that bile itself when imprisoned in the gallbladder by obstruction of the cystic duct, may set up such an inflammation in the gallbladder wall as to produce a characteristic pathologic picture of cholecystitis.

We were able to demonstrate that in dogs complete obstruction of the cystic duct did not produce inflammation of the gallbladder if the imprisoned bile is withdrawn and replaced by an equal amount of physiologic solution of sodium chloride. However, if bile were left imprisoned in the gallbladder and the cystic duct obstructed, inflammation of the gallbladder wall did take place and the severity of this inflammation was apparently in direct proportion to the nature and the concentration of the bile obstructed. Where this concentration was as much as one time actual necrosis of the gallbladder was frequent. If the obstruction of the cystic duct was not present, then the injection of such concentrated bile failed to produce any evidence of damage to the gallbladder wall. The substances in bile chiefly responsible for such injury appeared to be cholesterol or some of the bile salts. Of the latter group sodium taurocholate, sodium glycocholate and sodium desoxycholate were studied. The latter seemed to be the most damaging.

From these studies it seemed to us fair to conclude that three important factors were involved in the production of cholecystitis. These factors were obstruction of the cystic duct; the action of bile on the gallbladder wall; and the occasional secondary presence of bacterial infection which would be superimposed upon the chemically damaged tissue. While the bacterial infection is often so great as to overshadow all of the other factors, in all probability it is usually preceded by the chemical damage.

As most of the bile products that produced the changes in our experimental animals were related to cholesterol and cholic acid, it occurred to us that there might be some evidence available in the gallbladder wall of the presence of such material in human cholecystitis. Since many of these substances are lipoid in character it might be possible to distinguish such substances in the gallbladder wall. The most common infectious agents in cholecystitis belong to the streptococcus and colon groups of organisms and one would, therefore, expect a characteristic pyogenic reaction where infections were predominant. A reaction to lipoid material, on the other hand, would consist generally of edema, fibroplasia and a histiocytic response either as inflammatory cells, xanthoma cells or giant cells.

In a previous publication[2b] we called attention to the existence of such an inflammatory reaction to lipoids in damaged human gallbladders as was sought for above. In the present study we wish to expand these findings further.

Three hundred fifty-four gallbladders which were removed consecutively at the Barnes Hospital, from January, 1939, to August, 1943, are included in this study. Blocks of tissue were taken from the fundus and neck of the gallbladder. The tissue was fixed for the most part, in 10 per cent formalin solution within a few minutes after it was removed from the patient. The sections were cut and stained with hematoxylin and eosin. Occasionally Foot's modification of Masson's trichrome stain was used. Where microscopic evidence of lipids was seen both scharlach R and sudan III stains were

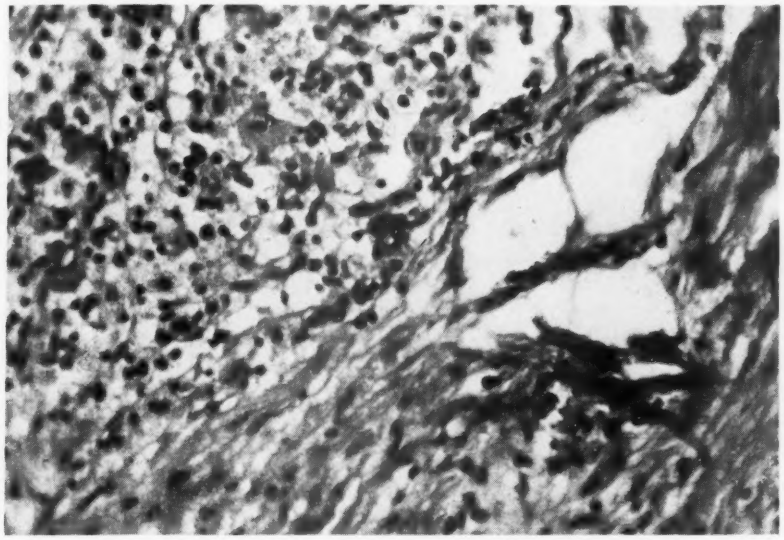
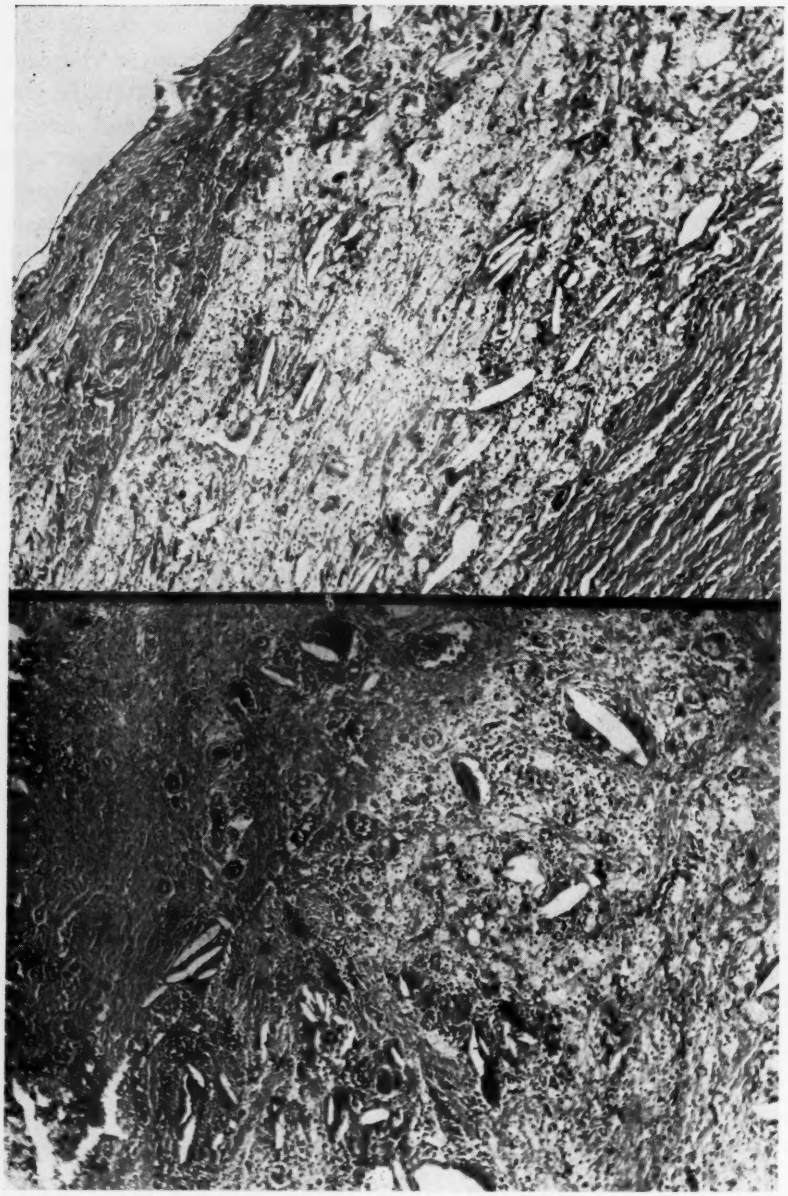


FIG. 1.—Histiocytes filled with what is apparently cholesterol ester and located deep in the wall of the gallbladder. Note the dense fibroblastic reaction.

used. Where it was thought necessary to use polarized light the material was previously prepared by incubation from three to six hours in digitonin.[3]

In 36 cases, or 10.2 per cent of our specimens, lipid material resembling cholesterol ester was found in histiocytes beneath the mucosa giving the characteristic picture of strawberry gallbladder. This, as a rule, was associated with but little damage to the gallbladder wall and was in most instances found in conjunction with cholesterol gallstones. In 80 cases, or 20.6 per cent of our material, there was microscopic evidence of either lipid or bile deep in the gallbladder wall. In almost every instance these gallbladders were badly damaged, most of them showing evidence of recent acute inflammatory reaction. Including cholesterolosis or strawberry gallbladder, therefore, this gave a total of 116 cases, or 32.8 per cent of the gallbladders removed, showing some histologic evidence of lipid or bile reaction in the wall.

An effort was made to identify the type of lipid present by histochemical means. There was, however, such considerable variation in the staining reactions that accuracy in the identification of the lipids was questionable In some of the instances edema of the gallbladder wall seemed to be partly



(a)

(b)

FIG. 2.—(a) Intracellular and extracellular cholesterolosis of the gallbladder wall. The latter apparently exists here as free cholesterol.
(b) What is probably an older phase of the same type of reaction as that shown in 2a. Note the increasing fibrosis and the giant cell reaction around the cholesterol crystals.

responsible. At other times the reason was not obvious. Thus, while we are considering most of these lipids as a form of cholesterolosis, it must be borne in mind that there is a possibility that some of them may not be of cholesterol origin. This, of course, is taken for granted where what is apparently desiccated bile is found. Certainly some of the lipids had their origin from red blood cells as could be determined by adjacent stainable iron in the tissue.

Perhaps the most common finding was that of lipid esters, presumably cholesterol esters in the cytoplasm of histiocytes resembling those found in the submucosa in strawberry gallbladder. Figure 1 illustrates such a finding. The cells present a granular, vacuolated or reticulated cytoplasm with a round or kidney-shaped nucleus in an eccentric position. The cells tend to be arranged in no particular architecture but exist as a focal conglomerate in a sheet-like form. In the fresh gross specimen these could often be seen as small yellow bodies, not a great deal unlike those seen in the strawberry gallbladder.

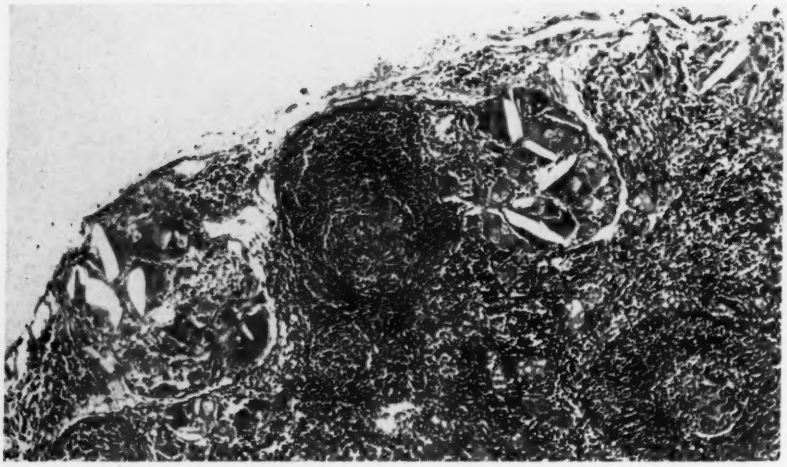
As a general rule, there was a considerable amount of fibroplasia generally seen around such areas of xanthomatosis. Where there was no evidence of extracellular accumulation of lipids the tissue reaction was generally not so acute. One of the most common of such extracellular substances apparently was free cholesterol. This is illustrated in Figures 2a and 2b. In these gallbladders one sees the same type of xanthomatous reaction as observed in Figure 1, along with slits and crevices representing spaces formerly occupied by what apparently were cholesterol crystals. Around such spaces one sees a giant cell reaction and lymphocytic invasion. Such a picture can be interpreted as a reaction of tissue to free cholesterol and to its esters.

It was not an unusual finding to encounter deposits of what apparently was free cholesterol in the gallbladder wall not associated with a considerable xanthomatous reaction. Figure 3a illustrates such a lesion. It will be noted that the deposit is just external to the musculature in the subserosa. This was not an infrequent location of this material. Such deposits, in fact, were probably more frequently encountered in the subserosa than in the submucosa. One of the interesting features about this particular specimen was the metastasis of the cholesterol to the regional lymph node at the junction of the cystic and common ducts. This is illustrated in 3b. These regional lymph nodes were not examined frequently enough microscopically to allow any statement as to how often such a phenomenon occurs. We observed it in only two instances in this series.

Xanthomatosis also often occurred in association with clear, irregular deposits of a brilliant orange-colored substance having all of the characteristics of the color of bile. This we have considered an extravasation of bile into the gallbladder wall. Where it was found associated with xanthomatosis the tissue reaction was often violent. Figure 4a shows such a reaction. In the upper right corner one may see the deposits of bile existing as dark, homogeneous, gray areas surrounded by histiocytes. At the lower left the serosa is edematous and contains a considerable number of polymorphonuclear leukocytes. This section is taken through the serosa and subserosa. Figure 4b gives an older reaction of a similar type taken through the gallbladder wall near the serosa in another specimen. The bile has become much darker in color and the inflammatory reaction shows a much older type of fibroplasia.

What apparently are deposits of bile may occasionally be encountered in the gallbladder wall without an associated xanthomatous reaction. Figure

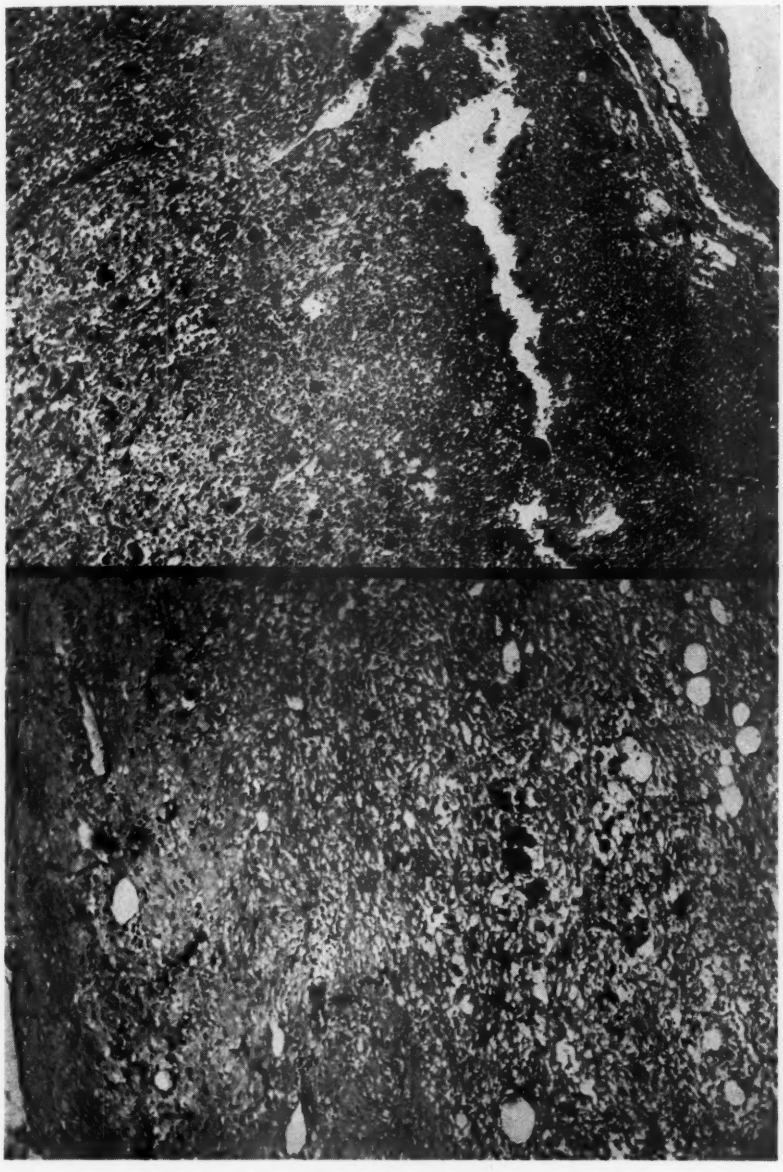
(a)



(b)

FIG. 3.—(a) Free cholesterol in the wall of a gallbladder with very little xanthomatous reaction and no edema. This is an old process.

(b) Regional (sentinel) lymph node removed along with gallbladder shown in Figure 3(a) demonstrating metastasis of the cholesterol through the lymphatics.



(a)

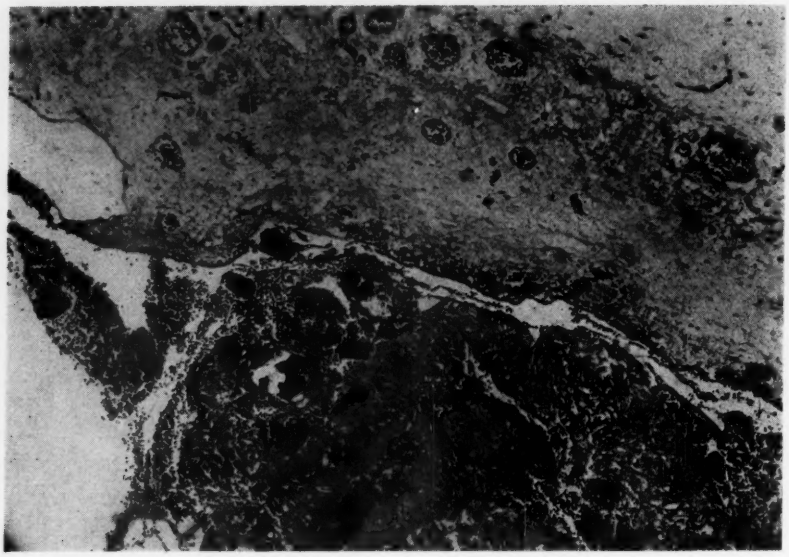
(b)

FIG. 4.—(a) Deposits of what is apparently bile may be seen in the left of the section surrounded by foam cells. Note the fresh exudate around the serosa.

(b) An older phase than that seen in Figure 4(a). The bile stains deeper, fibrosis is dense and fat replacement is beginning.

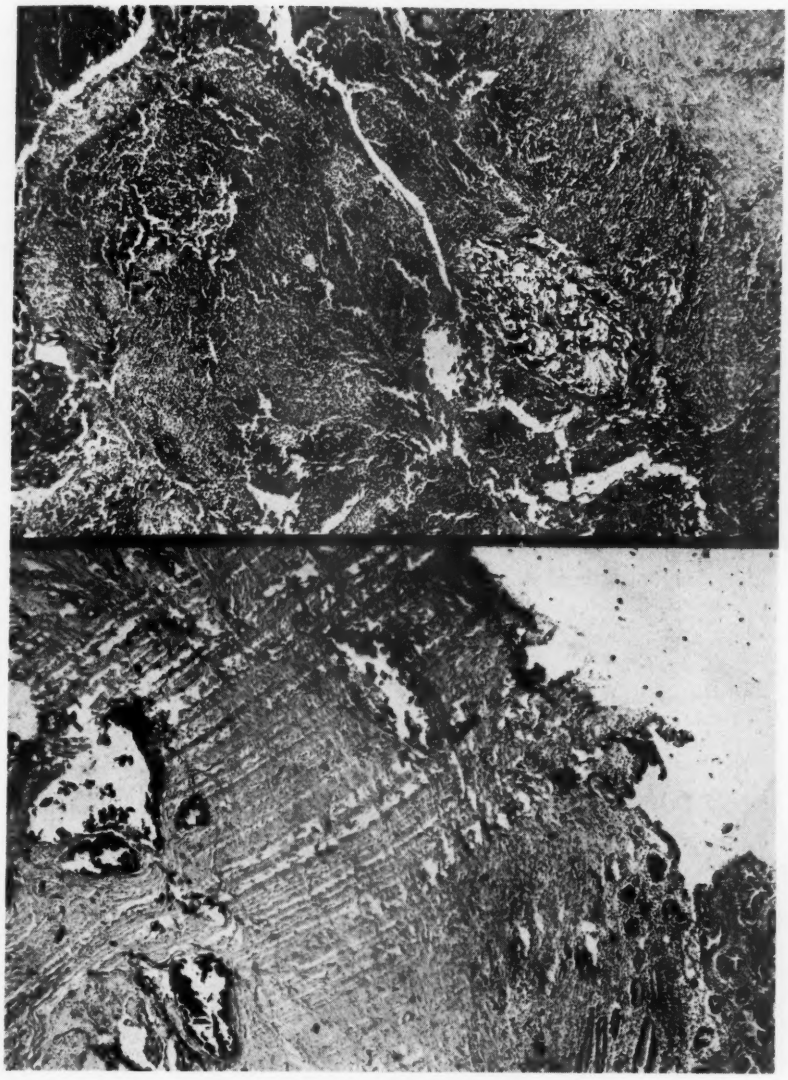
5a shows the appearance of such inspissated bile on the mucosa of the gallbladder. It should be noted that the adjacent epithelium is destroyed. Figure 5b shows such deposits of bile in an inflammatory exudate around the serosa of a gallbladder in which there are areas of old blood. In the stained section there was marked contrast between the different types of pigment, the bile being a brilliant orange and the blood a brick red.

(a)



(b)

FIG. 5.—(a) Deposits of inspissated bile on the mucosa of the gallbladder. Note the destruction of most of the epithelium.
(b) Deposits of bile in a region in which there is old blood. Note the difference in their characteristics.



(a)

(b)

FIG. 6.—(a) Large deposit of bile and cholesterol in the wall of a badly damaged gallbladder.

(b) A still older phase of bile deposit in a dense, avascular gallbladder wall. What seemed to be calcium salts were beginning to appear in the deposits.

Figure 6a shows an older process in which one sees a deposit of bile pigment in which there apparently was a considerable amount of cholesterol. The deposit is surrounded by a very dense inflammatory reaction consisting mostly of fibrosis and histiocytes. Figure 6b shows a much older phase of the same type of reaction. Here one sees several deposits of bile, or what is apparently bile as determined by its color, which have become so hard that the microtome knife was injured, evidence of which can be seen in the section. The fibrous tissue around such deposits is rigid. It is not unlikely that calcium

soaps are deposited in such areas and such a reaction may explain much of the calcification seen in the gallbladder wall on rare occasions.

Figure 7 is offered as evidence that the bile reaches the gallbladder wall by extravasation in some way. The lower portion of the section represents a cyst the wall of which is lined by scar tissue. The cyst contained pigmented

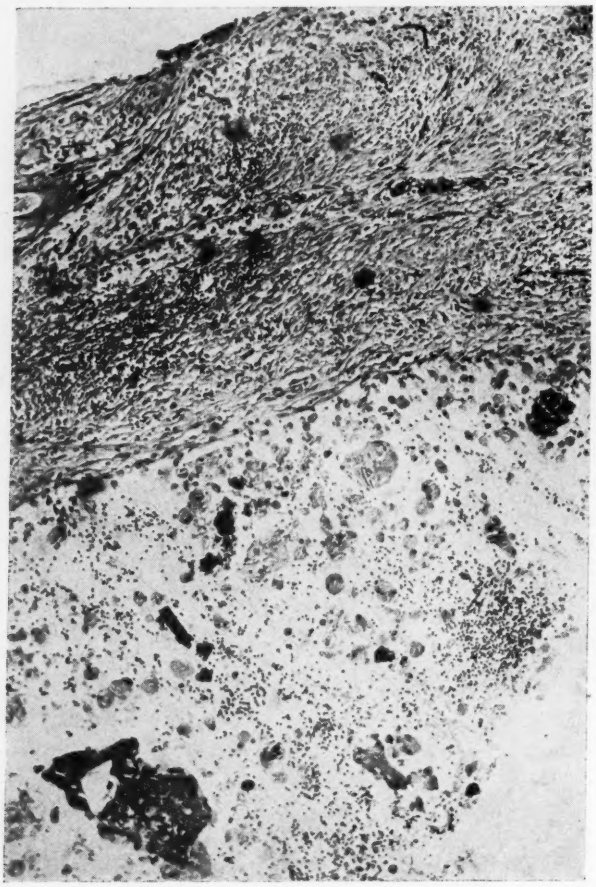


FIG. 7.—Cyst formation in gallbladder wall. The cyst is lined by scar tissue and contains bile elements.

material resembling bile and the microscopic appearance of some of the precipitate is identical to that described in previous sections.

Figure 8 is taken from another specimen showing what apparently is the crystallization of cholesterol taking place in the wall of a gallbladder. The striations are easily observed and the beginning giant cell reaction is obvious.

SUMMARY AND CONCLUSION

Since from experimental evidence most instances of cholecystitis have their beginning as a result of chemical injury to the gallbladder wall and since the

most likely chemical producing this injury is bile, gallbladders removed at operation for clinical cholecystitis were studied microscopically for evidence of bile or cholesterol in the wall. Three hundred fifty-four specimens removed consecutively for gallbladder disease were studied. Of these 116, or 32.8 per cent, showed some evidence of lipid or bile reaction. In this group is included the so-called cholesterolosis or strawberry gallbladder of which there were 36, or 10.2 per cent. In the remaining 80 cases, or 22.6 per cent of the material studied, there was easily demonstrable in the gallbladder wall evidence of tissue damage due to the presence of what was apparently cholesterol,

FIG. 8.—Crystallization of cholesterol in gallbladder wall suggesting that it enters the intercellular spaces in fluid form.

cholesterol esters or inspissated bile, itself. These substances were almost universally found associated with severe damage to the gallbladder wall. In no instance were they noted deep in the wall in the absence of severe tissue reaction. Some of the most characteristic microscopic pictures are described in detail. These data are offered as additional evidence that bile plays a dominant part in the production of cholecystitis.

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DISCUSSION.—DR. RAYMOND P. SULLIVAN, New York City: Doctor Sanders very briefly referred to diseased conditions of the common bile duct, which may be manifested in various types of trouble not only to the patient, but also to the surgeon. I refer particularly to strictures and tumors of the common bile duct, which demand reconstruction so as to insure a proper flow of bile into the intestinal tract. I now show you some lantern slides of cases illustrating some of these difficult and various conditions.

The first slide concerns a woman, age 57, admitted to St. Vincent's Hospital, New York City, in May, 1920, because of manifest signs of acute intestinal obstruction. At operation the intestinal obstruction was found to be due to a large gallstone impacted in a section of the jejunum. The passage of the gallstone was *via* a pathologic cholecystenterostomy. Removal of the gallstone corrected the obstruction. The internal fistula

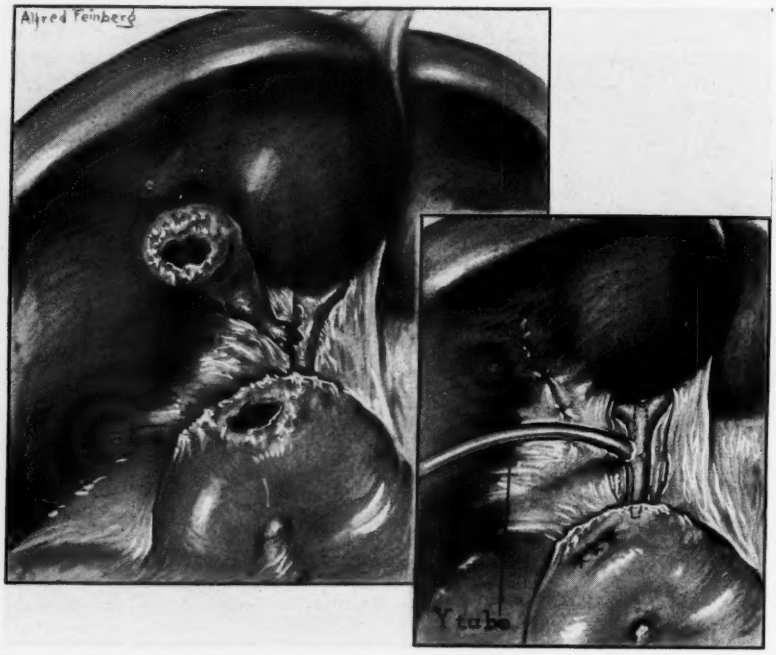

FIG. 1.—Represents the traumatic rupture of the common duct during cholecystectomy. Reconstruction over a Y-tube. Satisfactory result.

demanded repair, which was accomplished by separation of fundus of gallbladder from intestine. Repair of the intestinal opening was simple. Cholecystectomy was complicated by excessive traction on cystic duct sufficient to cause a rupture of the common duct, as demonstrated by the drawing. Immediate repair of this common duct was made by splitting a No. 16 F. rubber catheter for a distance of 2.5 inches; then introducing the limbs of the resultant Y-shaped rubber tube—in both upper and descending direction of the common duct—followed by closing the duct over the rubber tube by interrupted sutures. This worked well. Additional small rubber tissue drain placed in subhepatic area was removed in three weeks with satisfactory relief of all symptoms. Follow-up examination, in May, 1935, demonstrated freedom of any symptoms referable to bile ducts.

The second slide shows the case of a woman, age 33, who had a biliary fistula following cholecystectomy by another surgeon in July, 1931. The fistula resulted from injury to the extrahepatic portion of the common duct at the original operation. The procedure, as demonstrated herewith, was to mobilize the fistulous tract, into which a filiform bougie had been introduced, and to visualize the supraduodenal portion of the common duct. It was thus learned that a complete fibrosis of most of the common duct existed. Accordingly it was necessary to construct a new channel for transmission of bile flow. This was done by making a V-shaped full-thickness flap of the anterosuperior wall of the duodenum; then attaching the apex of the flap to the extrahepatic remnant

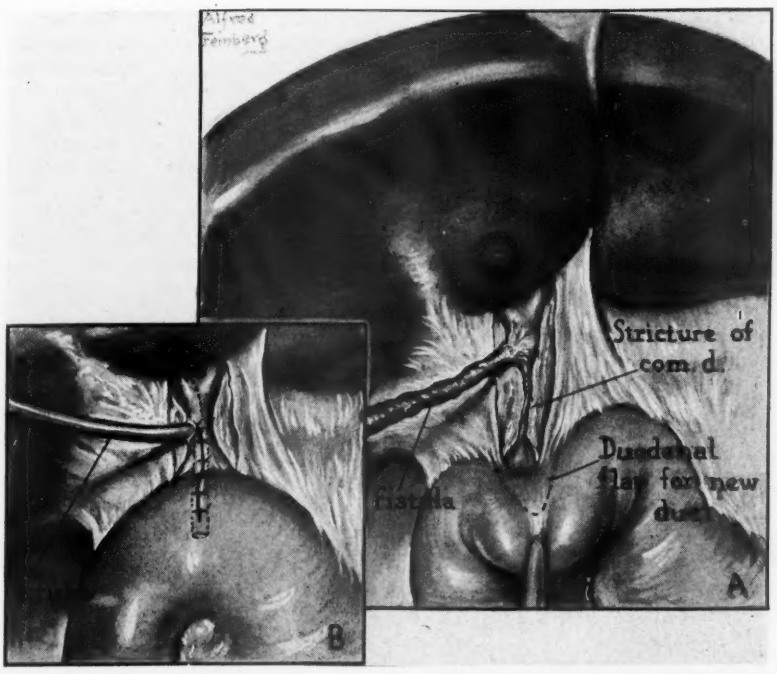

FIG. 2.—Case of biliary fistula necessitating reconstruction of common duct by full-thickness V-shaped flap from duodenal wall.

of the common duct; placement of a T-tube, and reconstruction of the wall of the duodenum over the limbs of the rubber tube. Secondary support was obtained by closing the periductal tissues over the reconstructed common duct. The result was satisfactory for two years when, following an attack of influenza, jaundice recurred, evidently the result of cholangitis and secondary stricture, as demonstrated by the drawings. Secondary operation revealed a stricture at the site of anastomosis between the duct and the duodenal wall. This area was excised and an indwelling section of a No. 16 F. catheter was used to reconstruct the common duct. The indwelling rubber tube was held by

FIG. 3.—Represents secondary stricture at the site of anastomosis between duodenal flap and common duct. Total excision of stricture over indwelling rubber tube, in residence two years.

interrupted silk sutures, with a completely satisfactory result. She was last seen in April, 1943, with no evidence of recurrence of jaundice or common duct symptoms.

The next patient, R. C., a man, age 45, was seen in August, 1937, because of jaundice, weight loss and severe colic. He had had no previous operation. However, he was "gassed" in World War I, and did not suffer from colic followed by jaundice until one year ago. In the past three months the jaundice was persistent and more intense. Operation, August 30, 1937, revealed a thick-walled gallbladder filled with stones and a tumor involving the gallbladder, cystic and common bile duct. The extrahepatic portion of the duct was considerably distended while the supraduodenal portion of the common duct was contracted. Cholecystectomy and removal of the entire cystic duct and the segment of common duct was performed, a T-tube was placed and the common

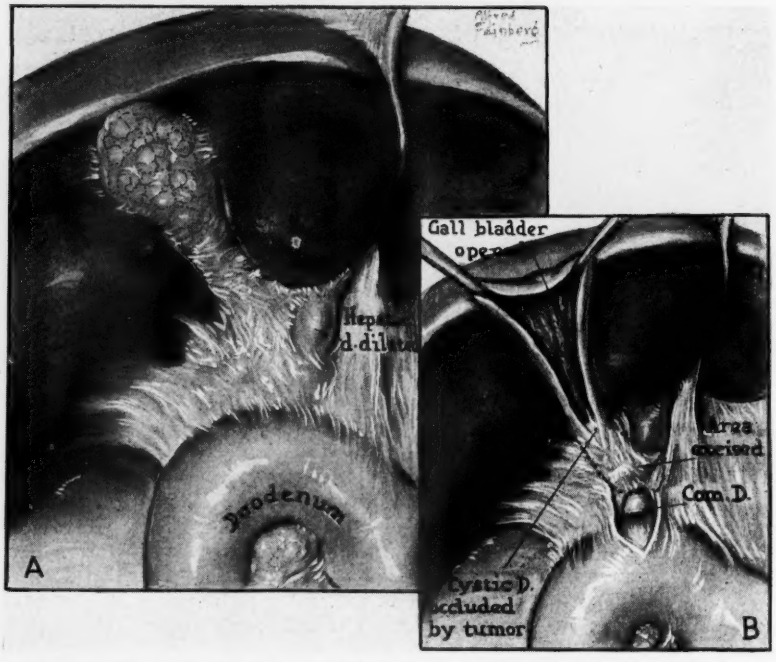

FIG. 4.—Represents the tumor of the common duct, associated with cholelithiasis. Involved section of the common duct removed with the gallbladder, in continuity. Reconstruction of common duct over T-tube, in residence eight months.

duct reconstructed using silk sutures. The subhepatic rubber tissue drains were removed on the sixth postoperative day. The T-tube was left *in situ* for eight months. The patient opened the tube every few days to observe the bile flow. He went back to work and the jaundice cleared satisfactorily within two months. Subsequent injection of lipiodol failed to reveal any evidence of stricture or tumor in the biliary tree. Accordingly, the T-tube was removed at secondary operation and an indwelling soft rubber tube was placed in the duct and held with chromic gut sutures. The result has been most satisfactory, and the patient is now serving as an infantry officer in Italy.

The next slide concerns a female (L. S.), age 42, who had had a cholecystectomy and choledochotomy for chronic suppurative cholecystitis and cholangitis in September, 1929. Recurrent symptoms brought her to the hospital in November, 1929. Operation revealed massive adhesions in subhepatic region, the area involved in the previous subhepatic abscess. These dense adhesions were firm enough to constrict the common duct at the site of the previous choledochotomy. This area, as shown by slides, was excised and a soft rubber tube was placed in the common duct and held by silk sutures. The result was very satisfactory, and to date there have been no symptoms. The rubber tube passed out after 14 months residence in the common duct.

The next slide concerns a woman who had had cholecystectomy for calculus disease

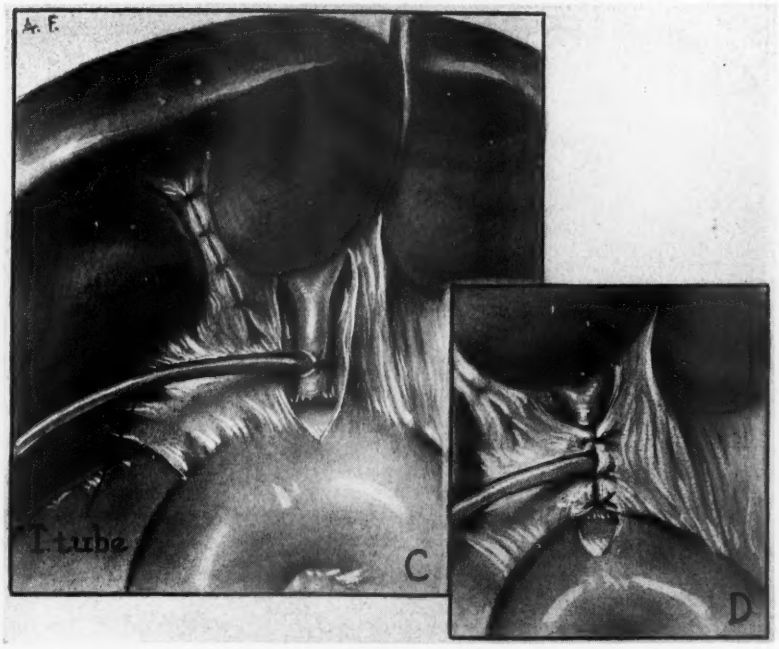

FIG. 5.—Removal of T-tube and placement of indwelling soft rubber tube to repair the defect following removal of T-tube.

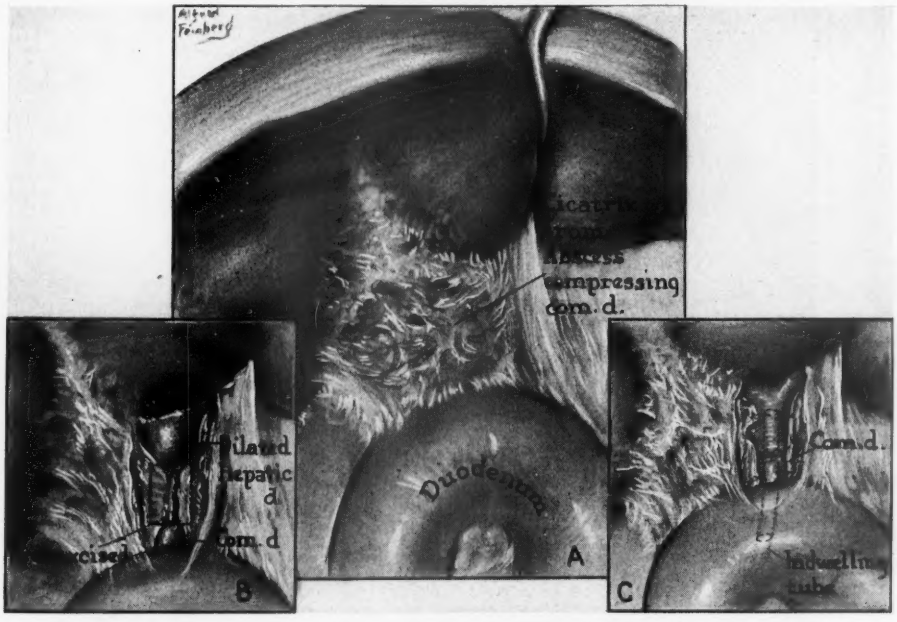

FIG. 6.—Represents stricture of the common duct caused by massive adhesions following cholecystectomy, complicated by subhepatic abscess. The strictured area was at the site of the choledochotomy. Excision of the stricture and reconstruction over indwelling rubber tube.

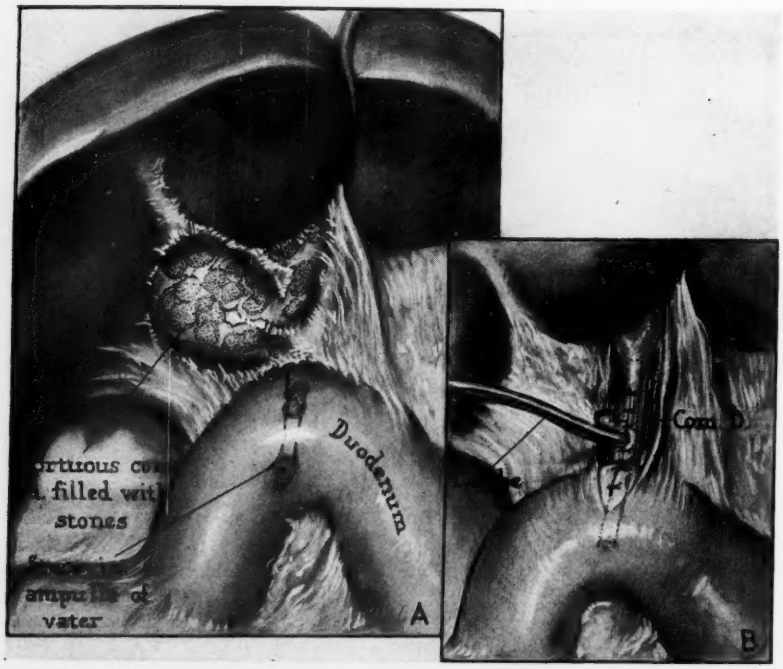

FIG. 7.—Represents the redundant common duct filled with stones. Stones also in the intraduodenal portion of the duct. Cholecystectomy 29 years previously. Redundant section of the common duct excised and the duct reconstructed over T-tube.

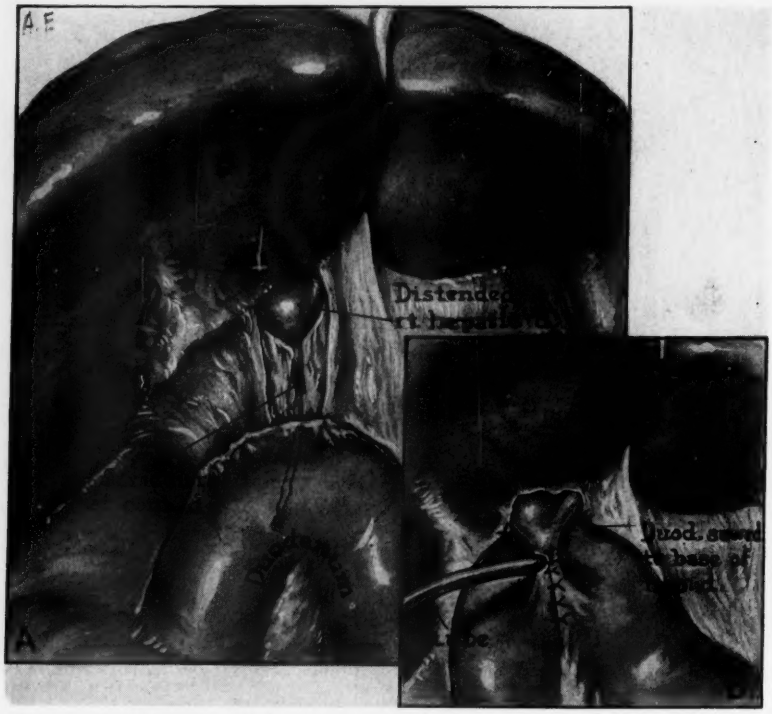

FIG. 8.—Reconstruction of the common duct following removal of T-tube (FIG. 7.). Reconstruction of the common duct over indwelling rubber tube.

29 years before our examination in February, 1942. At this time she was jaundiced and suffered recurrent attacks of colic, chills and fever, with weight loss. Operation February 23, 1942, revealed a loop of small intestine adherent to the scar of previous operation in such manner as to form an aperture through which another loop of small intestine was adherent, producing a recurring intestinal obstruction. When the condition was corrected, there was revealed a mass located in the liver sulcus which at first resembled a contracted gallbladder, but when mobilized was found to be an enlarged and redundant common bile duct packed with calculi. Stones were found at the ampulla of Vater and in the intraduodenal portion of the common duct and in both left and right hepatic ducts. The redundant portion of the duct was excised and the calculi removed; this necessitated dividing the intraduodenal segment of the duct. T-tube drainage was instituted for three months and then an indwelling tube of rubber was placed in the duct. The result was satisfactory; the jaundice disappeared, she gained in weight, and was symptom free. A small duodenal fistula persists with frequent passage of small concretions, but the patient, who is now age 70, is content.

The next case is that of a woman, age 34, who was seen in September, 1939, because of chronic duodenal ulcer and associated pyloric stenosis. During the mobilization of

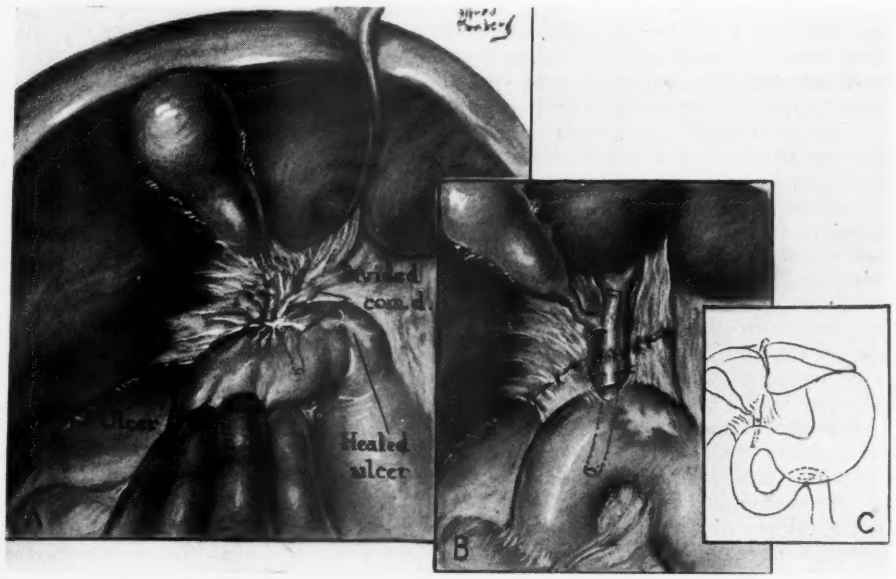

FIG. 9.—Necrotic destruction of the common duct caused by penetrating duodenal ulcer. Reconstruction of the duct over indwelling tube and posterior gastro-enterostomy. Residence of tube three years. Secondary removal of tube necessary because of impossibility of tube to pass along the proximal loop of jejunum.

the duodenum in preparation for gastrectomy, a gush of clear bile flooded the operative field. It was recognized that the duct was involved in the ulceration, which had caused necrosis of the duct wall. The duct ends were evened up, an indwelling rubber tube was placed in the duct, followed by end-to-end anastomosis of the duct ends. The tube was anchored by interrupted silk sutures. A posterior no-loop gastro-enterostomy was performed after cautery destruction of the duodenal ulcer. Recovery was uneventful. The tube remained in good position for three and a half years, when it loosened and caused symptoms of colic. It was easily removed by transduodenal delivery. At this time it was observed that the common bile duct was in excellent condition. Retention of the tube during the interval offered less irritation than a metal tube might have caused.

These cases are taken from a personal series of 22 cases of acquired stricture of the common duct in which there was but one fatality. The variety in this series supports the formula of great variability in the pathologic and anatomic conditions encountered, and hence the variety of operations necessary for repair of these diseased common ducts.

Dr. Charles Gordon Heyd, New York City: I think Doctor Sanders has reported a cross-section of the experience of most clinics in this country. In the series of our own which he so kindly mentioned, representing a 17-year period, one can see very definite trends in the type of surgery and indications for exploration. The group was made up of 54 surgeons. Quite obviously only the more demonstrable lesions were operated upon in the beginning. I should say we explore the common duct in about 14 per cent at the present time. There is an infrequent common duct condition that we have noted: A patient who has a degree or so of fever in the afternoon, has had a cholecystectomy some years ago, has an icteric index of 18 or 19, and on exploration is found to have a contracted duct. Drainage will reveal flocculent material, with sand, and drainage for a month or six weeks will bring about a cure.

The philosophy behind Doctor Sanders' paper is all-important. Biliary duct disease is a chronic disease and, without surgery, will end in chronic invalidism. We are apt to forget there are systemic manifestations of gallbladder disease long after the local symptoms have ceased to be the dominant factor in the patient's condition. Modern medicine has given us so many aids to ease a patient's disability that we are apt to make him comfortable at the expense of his future health and well-being.

Dr. Frank H. Lahey, Boston, Mass.: This is a subject in which we have been interested for a number of years, and it is an important subject because there is no doubt that family physicians frequently have been unhappy about the end-results of cholecystectomy for gallstones, and very properly so. This largely has been due to leaving stones in the common or hepatic duct.

We have established certain reasons for opening the common duct; dilatation of the common duct, contraction of the gallbladder, jaundice, suspected stone, induration of the head of the pancreas, and long-standing gallbladder infection with stones.

I would rather depend on the condition of the gallbladder than almost any other reason for opening the common duct. In other words, I open and explore the common and hepatic ducts when the gallbladder shows evidence of thickening or long-standing infection. At the Lahey Clinic we now open the common duct in 44 per cent of cases, which has resulted in the discovery of stones in 18 per cent. Our figures show that in 39 per cent of cases in which stones were found, no jaundice had occurred. For that reason, we have no confidence in limiting exploration of the ducts to those patients who are jaundiced.

Bile which is discolored and flocculent as it is withdrawn from the ducts is an indication for opening the ducts; however, stones sometimes exist in the presence of clear, yellow bile. Occasionally stones are present when the duct is not dilated. Therefore, one cannot set up clear-cut indications for opening the common and hepatic duct.

Too many common ducts are cut during cholecystectomy. We have operated upon 104 strictures of the common or hepatic duct, without ever having produced such a condition ourselves. Prevention of this accident involves a very simple principle. A gallbladder operation should not be undertaken without adequate anatomic exposure. There is no really completely satisfactory end-result after the common duct has been cut and there is a stricture. There are certain things that can be done, such as inserting indwelling tubes, vitalium or rubber. I recently published a report on one that had been in a hepatic duct for six years without symptoms. I do not like to put in a vitalium tube when a large section of the hepatic or common duct has been removed and the vitalium tube with its flange must rest directly upon the portal vein. I have sutured the portal vein too many times not to have a healthy respect for it. True, the portal vein has a low pressure, but it produces a high pressure in me when I cut it. When a large section of the common and hepatic ducts must be taken out, I would rather use a rubber tube. If I have to replace it in six or seven years, I can then insert a vitalium tube, lessening, thus, the possibility of penetration of the portal vein, as by that time the tube is surrounded by a good deal of scar tissue.

In the cases in which there is a large defect of the hepatic duct and no scar tissue remains in the bed left by the removal of the hepatic duct, it is an advantage to employ a T-tube since immediate decompression of the liver can be obtained. In these cases it is possible at the end of a year, when a good bed of scar tissue has surrounded the T-tube, to replace it with a straight rubber or vitalium tube and thus eliminate external biliary drainage.

As I have frequently said, I think the best treatment of stricture of the common or hepatic duct is better anesthesia, preferably spinal, plus better relaxation and exposure, with an accurate anatomic dissection about the junction of the common and hepatic ducts and the cystic artery. These precautions eliminate many postoperative problems.

DR. ROBERT L. PAYNE, Norfolk, Va.: The question of when to drain the common bile duct has been definitely laid down by Doctor Sanders and Doctor Lahey, but there has been no indication or rule laid down as to when a tube draining the common duct should be removed. Doctor Sanders merely passed this problem over by saying he removed the T-tube at variable periods of time. I wish to say that if we are going to lay down definite rules covering the institution of drainage of the biliary tree, then it would seem most reasonable that we should have some definite rules laid down concerning the cessation of tube drainage of the bile duct. In other words, we have been told over and over again when to put a tube in the common duct, but never when to take it out.

There are three factors tc be considered in removing a common duct drain: (1) The liver factor—function normal or depressed; (2) the duct factor—inflammatory cholangitis; and (3) the sphincter factor—sphincter spastic, inflamed or obstructed. Whenever the common duct is obstructed by inflammation, stricture, stone or new growth, there is an increase of the intraductal pressure, and when this exceeds the hepatic secretory pressure, liver dysfunction, as evidenced by incompetence, and often degenerative changes in the liver result. A common bile duct drain is so placed for the purpose of decompressing the liver and draining the inflamed ducts until a normal state is attained. It should further be maintained until obstruction at the sphincter of Oddi is completely overcome. The criteria for removing a common duct drain are based on (1) chemical study of bile; (2) microscopic study of bile; and (3) the determination of the patulence of the sphincter of Oddi.

In the presence of liver damage and cholangitis, hepatic bile obtained through the T-tube shows a decrease or absence of bile salts, a decreased calcium level, and an increase of chloride level. To simplify this chemical study for practical purposes, I use only the determination of bile salts and the bile chlorides. The chloride level of normal bile is the same as blood serum level; therefore, the chloride level must approach the normal 450 to 550 mg. per 100 cc. and the bile salts in normal quantity be shown before the chemical factor related to drainage is considered normal. Microscopically, the bile should be studied for crystals of calcium bilirubinate, mucus and pus. When these clear up, the microscopic evidence for draining the common duct is removed. The sphincter of Oddi must be open so that bile drains freely into the duodenum before the T-tube can be removed. The best methods of determining the patulence of the sphincter of Oddi is clamping the T-tube off for 12 to 24 hours and studying the blood for any increased serum bilirubin level and, in addition, retrograde injection of an opaque substance, like hippuran, through the T-tube, followed by roentgenographic visualization. I do not remove a T-tube until all three of these studies come together, namely, a normal liver factor, duct factor and sphincter factor, by which I feel that we have a more intelligent interpretation of when to take out the T-tube than simply to remove it empirically a certain number of days after its introduction into the common duct.

I realize that these methods described are distinctly crude and will permit of considerable improvement. I would urge that more investigation and study of this problem be made by all surgeons who are doing bile tract surgery, feeling that some day a more intelligent interpretation than I have presented will be offered as definite indications for discontinuing the drainage of the biliary tract.

DR. JAMES D. RIVES, New Orleans, La.: I would like to comment on two features of this discussion that seem to me pertinent. It is evident that in the country from which Doctor Lahey draws his cases the surgeons are less timid than they are in and around New Orleans. Here, we not infrequently see cases in which the surgeon has done an incomplete removal of the gallbladder or has left a very long cystic duct stump. Within the last year I have seen three such cases in which the remnant of gallbladder or duct caused recurrent attacks of jaundice in the absence of stones.

I would like to report a single case that seems to have bearing on Doctor Womack's suggestion that cholesterol may be deposited in the sentinel node. Several years ago I operated upon an elderly white male with complete obstruction of the common bile

duct and a long-standing history of chronic cholecystitis, with recurrent acute attacks. On exploration, I found a huge mass in the gastrohepatic omentum obscuring the common duct, the hepatic artery, and the portal vein. This was surrounded by very extensive subacute and chronic inflammatory reaction. I had at first thought that this was a huge stone that had been extruded from the common bile duct. By very cautious dissection, I was able to demonstrate that it lay entirely outside the duct and consisted of what appeared to be, and what on examination proved to be, a large mass of cholesterol, measuring about 2 x 3 x 4 centimeters. The common duct was then opened and several stones removed. No evidence of damage to the wall of the duct existed and I have concluded that this cholesterol deposit was probably in the lymphatic structures.

Dr. Warren Cole, Chicago, Ill.: I was much interested in Doctor Womack's statement that disease of the gallbladder is due to chemical factors and not primarily to bacteria, *i.e.*, the bacteria get in secondarily. We have arrived at the same conclusion experimentally. In a series of dogs we obstructed the cystic duct partially and observed the gallbladder for several months. For three months nothing happened to the gallbladder wall; however, after six months the wall becomes thickened. Microscopically, we found the same condition we see in the surgical pathology room, namely, thickening of the wall, fibrosis, *etc.* I believe Doctor Womack's explanation of cellular reaction to cholesterol and bile salts sounds very feasible. We all know that cholesterolosis has not shown evidence of being the cause of many symptoms. Nevertheless, his suggestion that cholesterolosis and deposit of bile may be an early stage in the pathogenesis of gallbladder disease appears as definite evidence that pathologic changes may develop.

Dr. Barney Brooks, Nashville, Tenn.: The remarks which have been made concerning the most appropriate time to remove a T-tube from the common bile duct prompt me to suggest that in my opinion it is best not to use a tube at all. We have not placed any sort of a tube in the common bile duct for a period of at least 15 years. I believe the only indication for the use of a T-tube is in instances in which this is the only method of establishing continuity between the hepatic duct and the intestinal tract. In other words, we use a T-tube only when there is not a common duct in which to place it. In instances in which a stone is removed from the common duct, the opening in the duct is carefully closed. A cigarette drain and small rubber tube are placed down to the site of closure, and in at least half the cases the patient recovers without any biliary fistula.

Dr. R. L. Sanders, Memphis, Tenn. (closing): Referring to Doctor Brooks' statement, a good many surgeons do not use drains in the common duct, but close the duct primarily after taking out the stones. I shall not argue this point, as I believe that whether one should or should not drain the duct is a matter of individual opinion and end-results.

As to anesthesia, we have used a general anesthetic, with a supplementary field block if necessary, in practically all our cases and have found relaxation excellent and exposure of the gallbladder and ducts ample. Fortunately, we have never injured nor accidentally cut the duct, so far as we know, nor have we encountered anything like the number of damaged ducts observed by Doctor Lahey. Certainly, we have no reason to alter our practice in regard to anesthesia, although we realize that the choice in this matter, also, is largely one of individual preference.

In common with many other members of this Association, for a number of years I have been teaching medical students in preparation for private practice. It is my opinion that we should be cautious in advising these inexperienced young men to open the ducts unless the indications are definitely present. Surgeons in larger medical centers, who have had many years of experience, might perform a choledochotomy in every other case without serious risk to the lives of their patients. The procedure is not without danger, however, and for the average surgeon, especially the one recently out of training, I consider such a practice contrary to good, sound surgical principles. From an experience including approximately 2,000 gallbladder operations over a period of more than 30 years, I am inclined to agree with Doctor Heyd that definite indications for opening the ducts are encountered in only 15 to 25 per cent of cases. Our end-results have proved the truth of this point.

SYMPOSIUM ON VASCULAR SURGERY

COMBINED VASCULAR AND NERVE INJURIES OF WARFARE*

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IT WOULD APPEAR that in this war the incidence of vascular injuries and of concomitant nerve injuries would probably surpass that of any other war. In addition to ordinary wounds produced by machine gun, rifle bullets and shrapnel, a great many multiple injuries are being produced by fragmentation of land mines, grenades and aerial bombs, as will be shown in a review of individual cases. Land mines and grenades may produce as many as fifty small individual wounds scattered over the body without causing death. This naturally increases the chance of vascular and nerve injuries. Of more significance to the study of major wounds to blood vessels and nerves is the fact that improved methods for the control of shock and infection have preserved a greater number of such injuries for subsequent observation.

Nerve lesions leading to motor and sensory paralysis of the upper and lower extremities are so obvious, because of their disabling consequences, that vascular injuries, at least in their early stages, are frequently overlooked. Conversely, vascular injuries in which a large pulsating hematoma is present may mask injuries to neural structures. With this in mind, it is of extreme importance that patients having nerve lesions be examined with great care lest an aneurysm or an arteriovenous fistula, or other blood vessel injury be missed. Only by a careful palpation, inspection, and auscultation of every wound can this be accomplished.

It is well to recall that all large blood vessels, with a few exceptions, are accompanied by peripheral nervous tissue. This is to be remembered especially in wounds of the neck where the great vessels lie in such close proximity to important cranial and cervical nerves. The same is likewise true in the brachial, femoral and popliteal regions, and while aneurysms of a traumatic nature are more frequently found at those points, it is nevertheless true that any vessel, regardless of its size or location, may be the seat of a disabling vascular injury.

It is not the purpose of this paper to review the common signs and symptoms of aneurysms, or of the ordinary manifestations of nerve injury or nerve section. Reports in the past which have dealt with aneurysms,

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have in the main neglected, or have failed to note concomitant nerve lesions. For this reason cases are presented here to show the frequent combination of such injuries.

In the inception of any vascular injury such as an aneurysm or fistula, external or internal bleeding may be severe and the immediate care, quite correctly, is directed to the control of the hemorrhage. In many instances, therefore, the appraisal of damage to neural tissue is delayed. The appearance of the external wound seen days or weeks after the original trauma is of little value in determining the state of the blood vessel or nerve injury. In many injuries hemorrhage may be slight, even though a major vessel is involved. In the absence of severe laceration, bleeding may be controlled by the pressure of the soft tissues and thus pulsating hematoma or false aneurysm will be formed.

The history will usually suggest the extent of the nervous tissue damage because even during the acute stage of the injury the patient, if conscious, is usually immediately cognizant of sensory and motor loss, whereas, the presence of an aneurysm or a fistula may be discovered only at a much later date. For instance, paralysis of a peripheral nerve recognized by the patient at the time of the wound might indicate severe trauma or even section of that nerve; whereas, if it came on at a later date in the presence of an aneurysm, it most certainly would be due to pressure, a fact of considerable prognostic importance.

In the presence of a large number of casualties and under battle conditions such as front line hospitals are now working, a diagnosis, though incomplete, or even incorrect, may be carried by a wounded soldier for some time. This does not imply that proper treatment has not been given. but it does entail upon those who are working in the Zone of the Interior, or in general hospitals distant to the battlefield, the necessity of particularly careful examination, uninfluenced by any previous diagnosis. Moreover, it is a well known fact that a vascular lesion may develop slowly to the point of recognition. For example, an arteriovenous fistula, surely present from the onset of injury, may not show the characteristic signs until some time later when edema and tissue hemorrhage have been absorbed. This is substantiated by the fact that vascular lesions were discovered in five patients in this general hospital upon whom only neurologic diagnoses had been made previously.

The neurologic sequelae of gunshot wounds of the central and peripheral nervous systems have varied but little in the previous armed conflicts in which these disturbances have been studied. The causalgia that Weir Mitchell described so graphically in the American Civil War differs in no respect from that observed today in Army General Hospitals. In this war the character of the inflicting forces has altered both the type and particularly the frequency of injuries to neural tissue as exemplified in the first instance by blast injuries and in the second by aerial bomb and antipersonnel land mine injuries.

I—VESSEL-NERVE INJURIES OF THE NECK

Among these previously well recognized neurologic syndromes are those extracranial lesions of the last four cranial nerves caused by gunshot wounds. As Pollock and Davis[1] have stated, the number of such syndromes is limited only by the possible combinations of complete or incomplete paralyses of these several cranial nerves and the descriptive ability of the various observers. Among the classical syndromes involving all or several of the last four cranial nerves are those of Avellis,[2] consisting of a unilateral paralysis of the soft palate and the larynx, from a lesion of the vagus and the internal branch of the spinal accessory nerve; of Schmidt,[3] characterized by the inclusion of the external branch of the spinal accessory nerve to the foregoing symptoms, and of Jackson,[4] which includes, in addition to all these symptoms, involvement of the hypoglossal nerve.

During the course of and following World War I, Vernet,[5] Collet,[6] Sicard,[7] Villaret,[8] Barbé,[9] Körner,[10] and Pollock[11] added significant contributions to the literature of War injuries involving these neural structures.

These authors gave to the combination of cranial nerves involved descriptive terms denoting either the neurologic distribution of or the anatomic location of the causative wound, such as, "syndrome of the posterior lacerated foramen" (Vernet), "glosso-laryngo-scapulo-pharyngeal hemiplegia" (Collet), "complete syndrome of the last four cranial nerves" (Sicard), and "syndrome of the posterior retroparotid space" (Villaret).

Whatever the syndromes may be termed, the resulting neurologic dysfunction depends upon the close anatomic continuity of the last four cranial nerves in the early part of their extracranial courses. In the jugular foramen, the glossopharyngeal, vagus and spinal accessory nerves, in order from before backward, lie between the inferior petrosal sinus anteriorly and the internal jugular vein posteriorly. At its exit from the jugular foramen, the internal jugular vein lies to the outside of the nerves and the carotid artery, with its accompanying sympathetic plexus, is placed closely in front and medially to these nerves. The hypoglossal nerve is in close proximity since it leaves the skull through the adjacent anterior condyloid foramen. In their more peripheral courses, the four nerves remain in close approximation to a point slightly below the tip of the mastoid, in the so-called retroparotid space. More laterally placed, in the coronal plane of the head, lie the seventh and fifth nerves.

Although reports of injuries to the jugular vein and carotid artery in association with injuries to the last four cranial nerves are rare in the World War I literature, such a combination of nerve and vessel injury must have occurred with some frequency. Among these infrequent observations of similar cases is that noted by Heyrovsky[12] who described involvement of the last four cranial nerves with a spurious aneurysm of the vertebral artery. Aneurysmorrhaphy was successful, but as in Case 1 (*infra vide*) the neural defect showed no evidence of improvement. That they were not reported would indicate that these cranial nerves may be injured without concomitant

vascular injury, as noted by Vernet, or that such vascular injuries may occur and heal spontaneously, or that the vascular injury was so formidable that, lacking modern methods of shock control, these cases did not survive. It is obvious that grave vascular injuries of a chronic, progressive nature do occur with the various classical syndromes indicating involvement of the last four cranial nerves, and that, furthermore, the control of such vascular injuries is of paramount importance in the rehabilitation of the wounded soldier.

Case 1.—*C. J. H., Hosp. No. 991: Gunshot wound of face. Removal of .44 caliber bullet in left occipitomastoid region. Involvement of V, VII, X, XI and XII cranial nerves and production of A-V aneurysm. No improvement in neurologic status after resection of vascular lesion.*

On November 10, 1942, this soldier was struck in the left malar region at close range with a French .44 caliber revolver bullet. The missile traversed the face, the lateral aspect of the neck, and became imbedded in the soft tissues of the left occipitomastoid region. He staggered back under the impact of the bullet but did not lose consciousness. He noticed at once loss of feeling in the mouth and lower left side of the face. His voice was hoarse, and he was unable to fully lift the left arm from his side. There was no extensive external hemorrhage, but the tissues of the side of the neck swelled considerably. Within a few days he became cognizant of a loud buzzing in his left ear. Upon occasion, his pulse rate would become very rapid. On December 22, 1942, the bullet was removed from the left occipital region, and on February 22, 1943, the left common carotid artery was ligated, without change in his symptoms.

On admission to the Ashford General Hospital, two months later, there was a small scar over the left malar region, denoting the wound of entry. There was a short operative scar 4 cm. mesial to the tip of the left mastoid. The tissues in this region appeared full in comparison to those on the right. Palpation revealed a deeply situated pulsating mass with a distinct systolic thrill. Auscultation noted a continuous bruit accentuated in systole with maximal intensity over the occiput which could be obliterated by deep pressure over the sternomastoid muscle. Arterial pulsation was absent over the left common carotid artery, the site of the earlier ligation. It was thought that this was an arteriovenous fistula of the cirsoid type, probably involving the occipital artery and vein deep to the muscles and posterior to the mastoid process. The question of involvement of the vertebral artery with accompanying veins in this region was considered.

Neurologic examination disclosed the following findings of significance. The pupils were of equal size, and there was no enophthalmus. There was analgesia and anesthesia over the third branch of the left fifth nerve; there was loss of taste over the anterior two thirds of the left side of the tongue; the soft palate was pulled to the right upon phonation, and the gag reflex was absent on the left side; the voice was hoarse and laryngoscopy disclosed a left abduction palsy; the left arm could not be abducted above 90 degrees, and there was atrophy of the left sternomastoid and of the lower two-thirds of the trapezius muscles; the superior third of this structure was incompletely paralyzed. The left side of the tongue was atrophic and furrowed and protruded to the left (Fig. 1).

The neurologic diagnosis was involvement of V, special visceral afferent (taste) of VII, IX, X, XI, and XII cranial nerves, similar to those syndromes reported by Collet, Vernet and Sicard, with the addition of the V and VII nerve lesions. To this was added the diagnosis of an arteriovenous aneurysm, probably of the occipital vessels.

An excision of these vessels was carried out on June 15, 1943. A semicircular

incision beginning over the left mastoid muscle was carried upwards over the base of the skull to the midline. The deep muscles were cut and retracted caudally. The external carotid artery was ligated, but this had but little if any effect upon the cirsoid aneurysm, which could be seen and felt at the base of the skull. This aneurysm was excised by ligating and cutting numerous arteries and veins which were seen to communicate with vessels entering the skull. Considerable bleeding was encountered,

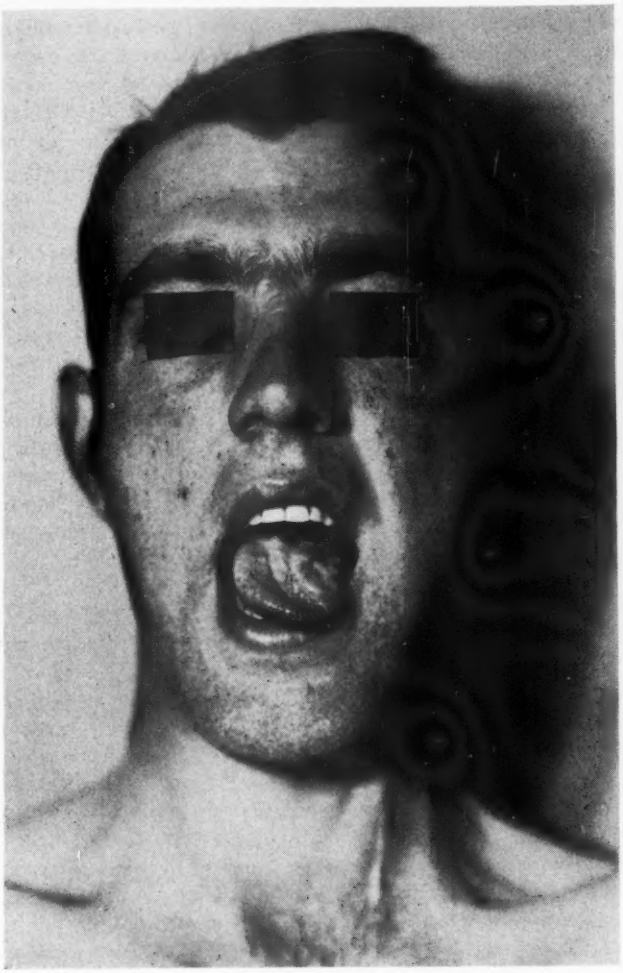

FIG. 1.—Case 1: Paralysis of left hypoglossal nerve.

which was controlled by coagulation and by the use of fine silk ligatures. At the end of the operation the bruit and thrill had disappeared.

Two weeks later, as a result of a friendly wrestling match, the wound was opened and a severe secondary hemorrhage occurred. This was controlled by hemostats which were left in place, and by packing. Following this the vertebral artery was ligated, which apparently controlled the bleeding since no further hemorrhage occurred with the removal of the clamps and packing. The wound healed uneventfully. There was no return of the bruit and thrill. There was no change in the neurologic findings upon discharge three months later and upon review examination three months after discharge.

Case 2.—*H. C. H., Hosp. No. 1397: Gunshot wound left side of neck, April 31, 1943. Involvement of cranial nerves VII, X, XI, XII, and second cervical with incomplete cicatricial occlusion of left common carotid artery. Artery freed from cicatrix. Progressive improvement in neurologic defect.*

On March 31, 1943, this soldier was injured by aerial bomb fragments which struck the left side of his neck, left thigh, and left frontal region of the scalp. He was unconscious for two hours. Severe hemorrhage persisted from the neck wound, and two plasma transfusions were given during the first 24 hours after the injury. Forty-eight hours later the neck wound was explored, and foreign bodies were removed. At this time he was unable to talk, move the left side of his face, or shrug the left shoulder. He noted loss of feeling over the lower third of the left side of the face. He had some difficulty swallowing fluids. Three weeks after the injury, the aphonia cleared, and his voice became progressively less hoarse. Both facial and shoulder movements improved slowly but steadily. From that period in his convalescence until his admission to Ashford General Hospital on May 10, 1943, he noticed an intermittent singing noise in the left ear.

There was a small, stellate, puckered scar below and anterior to the tip of the left mastoid. Palpation over the scar and in the anterosuperior cervical triangle disclosed a small, tubular expansile mass. Auscultation over the mass revealed the presence of a harsh, continuous bruit, accentuated in systole, which could be obliterated by compression of the common carotid artery.

Neurologic findings included an apparent enophthalmus on the left, moderate weakness of the musculature about the left angle of the mouth, normal taste perception and hoarseness with an abduction paralysis of the left vocal cord. The superior third of the left trapezius muscle was atrophic with weakness of shoulder elevation, but with normal abduction of the left arm. The tongue was atrophic and protruded to the left. There was an area of hypalgesia and hypesthesia corresponding to the anterior fibers of the second cervical nerve.

The neurologic diagnosis was involvement of the VII, X, XI and XII cranial and the second cervical nerves. With the exception of the VII nerve disturbance this case was similar to the syndromes previously recorded. The more inferiorly and laterally placed site of injury in this instance explained the lack of involvement of the glossopharyngeal nerve. Surgical exploration of the expansile mass in the cervical region disclosed a cicatricial, incomplete occlusion of the common carotid artery with proximal dilatation of the vessel. The neurologic disturbance, with the exception of the abduction paralysis of the left vocal cord, improved steadily until discharge three months later.

The release of scar tissue about the carotid artery led to complete disappearance of the bruit and thrill, a finding worthy of note, since apparently scar tissue contracting about a vessel may produce the cardinal physical findings of an arteriovenous fistula.

Case 3.—*R. Z., Hosp. No. 3007: Gunshot wound, 20 mm. shrapnel, left mastoid region on December 3, 1942. Involvement of cervical sympathetic, VII, IX, X, XI and XII cranial nerves, associated with A-V aneurysm of internal carotid artery and internal jugular vein and evidence of increased intracranial pressure. Resection of A-V aneurysm. Moderate improvement in neurologic status.*

This soldier was injured on December 3, 1942, by pieces of shrapnel from a 20 mm. shell which struck the left mastoid region and left shoulder. The former wound bled profusely for a few seconds, and then hemorrhage ceased spontaneously. Three days later the mastoid-cervical wound was explored and a foreign body removed. On the following day, he noted a buzzing sound in the left ear, the appearance of a swelling below the wound and for the first time, developed moderately severe generalized headache. There was weakness in the muscles of the lower half of the left side of the face, and his voice was hoarse. He noticed that choking would follow the ingestion of fluids, but that

solid food was tolerated without difficulty. When he became ambulatory, he realized that he could not abduct the left arm above 90 degrees. On April 3, 1943, the left common carotid artery and left internal jugular vein were ligated. On June 11, 1943, the A-V aneurysm involving these vessels was resected, both procedures being performed by Lt. Colonel William V. Cox, M.C. The patient's convalescence from these operations was without untoward incident. Neurologic examination was carried out in the Ashford General Hospital on August 30, 1943.

The site of the operative procedure showed nothing abnormal, and there was no evidence of recurrence of the arteriovenous aneurysm. Vision in the O.D. was 20/20, in the O.S. vision was reduced to hand movements, with fingers recognized in the

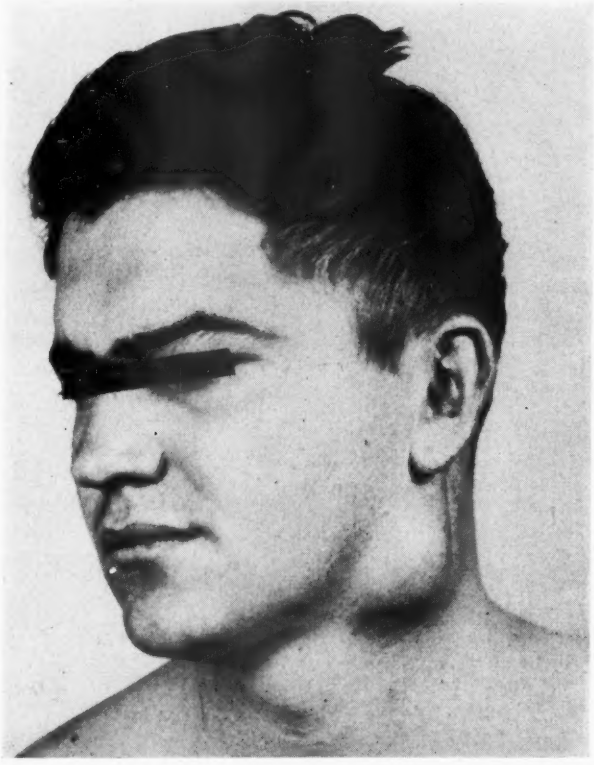

FIG. 2.—Case 3: Arteriovenous aneurysm of internal carotid-jugular vein. The vascular lesion and the narrowed palpebral fissure are apparent. (Case of Lt. Colonel William V. Cox.)

temporal quadrants. The optic nerve head of the right eye was normal. The left neuroretinal outline was clear, but the temporal half of the disk was pale. Adjacent to the temporal margin were two or three hyaline deposits and similar deposits were noted nearer the macula. An area just above the disk was pale. The changes in this fundus suggested an earlier papilledema with subsequent atrophy. Previous studies, prior to operation, had disclosed a bilateral papilledema of three diopters.

The pupils were equal and active to light stimulation and upon accommodation. There was intermittent conjunctival injection on the left. On July 9, 1943, Captain C. N. Eastman, M.C., had reported a narrowing of the left palpebral fissure, enophthalmus, some decrease in intra-ocular tension as compared to the right and normal side, loss of the ciliospinal reflex and loss of sweating over the left side of the face. There was

no change in sensation over the domain of nerve V. There was a contracture of some muscle fibers at the angle of the mouth, causing inversion of this structure. There was weakness of the risorius, triangularis and lower fibers of the orbcularis. There was slight loss of the normal palatal arch on the left and the soft palate pulled to the right upon phonation. The voice was hoarse and laryngoscopy revealed an abduction paralysis of the left vocal cord. The left sternomastoid muscle was functionless, and the left arm could not be abducted above 90 degrees.

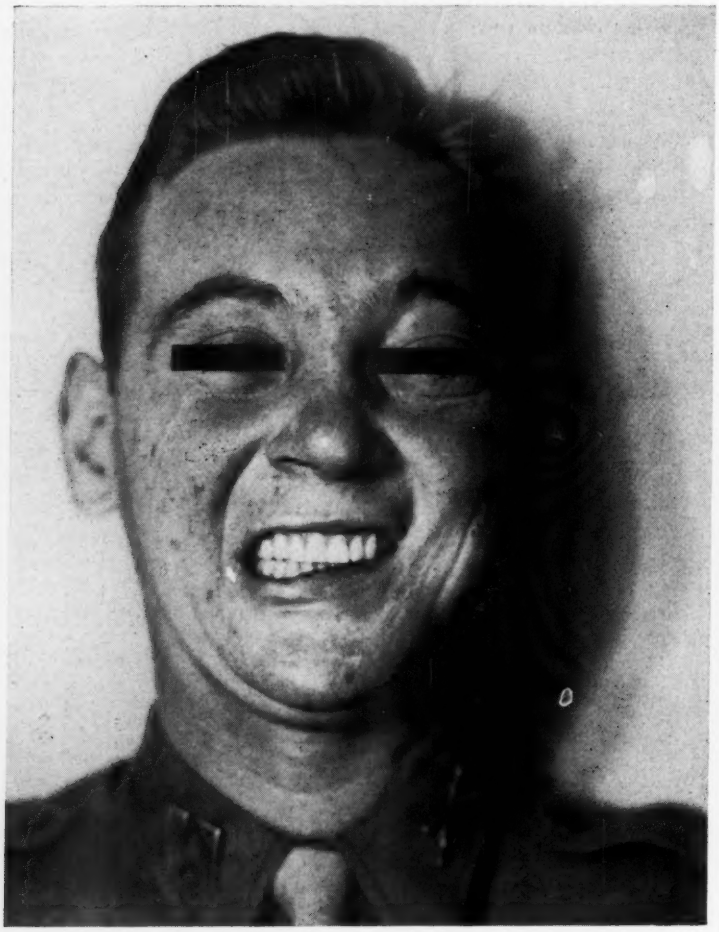

FIG. 3.—Case 4: Exophthalmos. Left seventh nerve paresis. Note wound of entry on lateral aspect of nose.

The neurologic diagnosis was incomplete involvement of the cervical sympathetic chain, and cranial nerves VII, IX, X, XI and XII, the syndrome described by Villaret. All components of the neurologic picture had improved considerably except those involving cranial nerves X and XI. Among six gunshot wounds of the last four cranial nerves described by Vernet with Lanvois and Patel is one quite identical with this case, but concerning which no postoperative course is available.

From the overseas clinical record it is clear that this patient had bilateral papilledema of three diopters before operation. This would suggest an increase in pressure in the venous circulation of the brain. Such a course is not incompatible with what is known

of the physiology of an arteriovenous fistula, where arterial blood at an increased pressure enters the venous system (Fig. 2).

Case 4.—*J. D. M., Hosp. No. 1345: Gunshot wound of face and neck. Involvement of V, VII, and XIth cranial nerves with development of arteriovenous aneurysm of cavernous sinus and internal carotid artery. Ligation of common carotid artery.*

On December 12, 1942, this officer was struck by a .30 caliber bullet. The missile entered 1 cm. mesial to the inner canthus of the left eye, and made its exit beneath the left mastoid region. He became unconscious at once and remained so for 24 hours. He was confused and drowsy for the following four days. Débridement was carried out on the evening of the injury, and sequestrectomy of a portion of the condyloid process of the mandible was done ten days later. He early appreciated numbness along

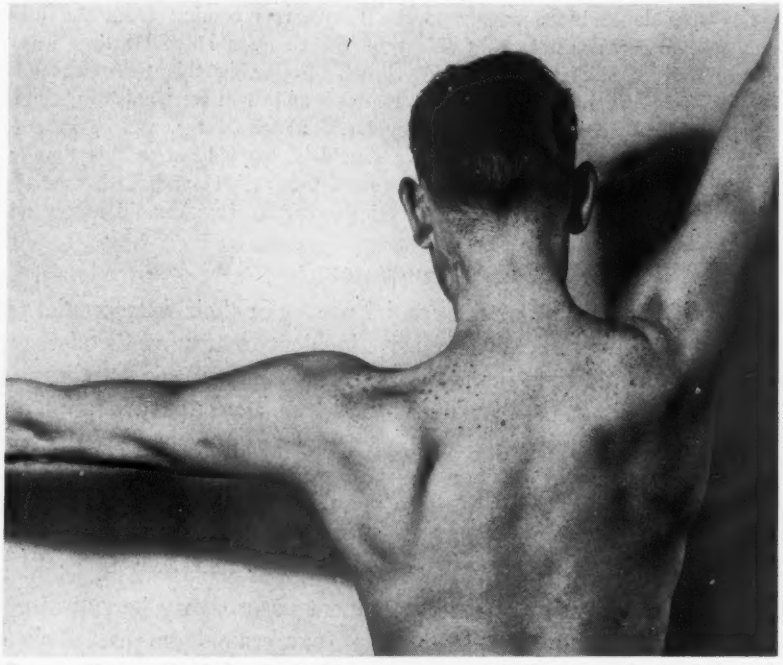

FIG. 4.—Case 4: Paralysis of spinal accessory nerve, left. Note wound of exit beneath mastoid region.

the lower half of the left side of the face, weakness of the left facial musculature and an inability to abduct the left arm above 90 degrees. From the time of adequate orientation, he had noted a protrusion of the left eyeball which had never diminished in size. Occasionally he reported hearing a "roaring sound like the ocean" in his left ear.

The striking feature upon first examining this officer was the moderate proptosis of the left eyeball. There was conjunctival edema and vascular congestion. There was a small, round scar on the lateral aspect of the nose 1 cm. from the inner canthus of the left eye. Below the tip of the left mastoid was a 2 x 4 cm. scar which was adherent to the deep structures of the neck.

Neurologic examination disclosed normal direct and peripheral vision. The left neuroretinal outline showed minimal blurring along its nasal margin. The retinal arterioles pulsated, and the veins were full. There was hypalgesia and anesthesia over the third branch of nerve V. There was almost complete paralysis of the circumoral musculature on the left side. There was pronounced atrophy of the sternomastoid muscle on the left. The trapezius muscle was atrophic with marked winging of the vertebral border of the scapula and loss of abduction of the left arm above 90 degrees (Figs. 3 and 4).

The supra-orbital veins were engorged, and a distinct, continuous thrill could be felt in both the upper and lower left eyelids. This thrill was transmitted into the neck over the carotid vessels. Over the left eye, forehead and left side of the neck there was a harsh, continuous roaring bruit, accentuated in systole. The left eyeball pulsated slightly. The whole picture was one of pulsating exophthalmos and was thought to be due to a communication between the cavernous sinus and the left internal carotid artery. On May 26, 1943, the left common carotid artery was partially compressed with an aluminum band, and it was felt that about 80 per cent of the blood flow through this vessel was stopped. Following this there was definite improvement in the exophthalmos, and, on June 23, 1943, the carotid artery was again exposed, together with its terminal branches. At this time temporary occlusion of the common, internal, and external carotid vessels did not obliterate the bruit previously described, and it was felt, therefore, that a large blood supply to the fistula was coming from the other side and from the vertebral vessels. At this time the common carotid artery was doubly ligated with braided silk and the wound closed. Following this there was additional improvement in the exophthalmos, although the bruit and thrill were only slightly affected.

The patient was seen in consultation by Dr. Rudolph Matas, and he agreed in the opinion that no further operative procedure should be carried out at this time.

The neurologic diagnosis was involvement of the V, VII and XIth cranial nerves associated with an arteriovenous aneurysm of the left internal carotid artery and cavernous sinus (Figs. 3 and 4).

DISCUSSION

Any specific syndrome of the cranial nerves in their extracranial courses depends upon the unpredictable vagaries incident to trauma through gunshot wounds and upon the time interval at which the neurologic observations are made. Although it is obvious that their close anatomic grouping sets the stage for multiple involvement of the last four cranial nerves, yet their separation into syndromes is of dubious neurologic import. From a study of these cases, and from other traversing wounds of the face and neck without significant change at the time of neurologic study, several observations of prognostic import seem worthy of comment. In the first place, injuries of nerves V and VII of transient or of rare permanent nature, may be engrafted upon the usual classical syndromes of the last four cranial nerves. This superimposed neurologic defect has exhibited a strong tendency toward spontaneous regression in the patients under observation. In the second place, a lesion of the last four cranial nerves, whether acute or of a chronic and perhaps regressive character, may incapacitate the patient in only two significant respects. In Case 3, there was pronounced difficulty in the early days after the injury, in swallowing fluids. Six months after the injury, the persistent abduction paralysis of the left vocal cord precipitated respiratory collapse at the beginning of induction of anesthesia, preliminary to the introduction of an intratracheal tube. A similar episode occurred in this hospital under identical circumstances in the treatment of Case 2. The involvement of the external branch of the spinal accessory nerve has shown little tendency toward improvement and the loss of abduction of the upper extremity above 90 degrees remains as a defect of considerable magnitude. Direct exploration of the region of the jugular foramen with the view of decreasing this neurologic disturbance has not been carried out or contemplated in these cases.

Finally, the experience that vascular injuries are associated with many injuries of the last four cranial nerves may not represent the true frequency of the combined injury, since this hospital has been designated as a Vascular Center. The combined injury, whether immediately fatal from hemorrhage or amenable to treatment, must be fairly common because of the anatomic contiguity of the vascular and neural structures.

The vascular lesion may include, in individual cases of a chronic nature, cicatricial occlusion, A-V aneurysm, false aneurysm or even similar lesions from indirect violence similar to Case 4. The fact that the vascular lesion in Case 2 was unrecognized previously is worthy of comment. The vascular injury may bear no relationship to the extent or duration of the neural damage except that of a concomitant injury. In these patients at least, the neural disturbances developed immediately following injury and were not favorably influenced by repair of the vascular lesion. That the aneurysmal mass may influence adversely the extent of neural injury in some instances is not disputed.

II—VESSEL-NERVE INJURIES OF THE UPPER EXTREMITY

It is the consensus of opinion of former writers on this subject that neural complications are much more common in aneurysms of the upper extremity than in those of the lower extremity. Thus Fromme,[13] in a long, carefully documented article, recorded 13 aneurysms of the upper extremity with neural changes out of a total number of 16. Makins[14] stated that 25 per cent of the subclavian aneurysms showed involvement of the brachial plexus and out of 54 cases involving the axillary and brachial arteries, 43 per cent showed involvement of neural structures. In Maurer's[15] series of 71 aneurysms of the upper extremity, approximately 25 per cent showed neural complications. On the other hand, in Makins' extensive series of 170 femoral artery injuries and 85 popliteal artery injuries, concomitant nerve injury was reported as rare. Illustrative of the combined vessel-nerve injuries to the first part of the axillary artery is that described in the following case:

Case 5.—*D. F. H., Hosp. No. 2869: Traversing gunshot wound of left infraclavicular space on March 28, 1943, followed by paralysis of left arm for two months. Subsequent resection of false aneurysm of first part of left axillary artery with slow improvement in neurologic status.*

On March 28, 1943, this soldier was struck by a rifle bullet in the left infraclavicular space which traversed the apex of the axilla and made its exit lateral to the tip of the left scapula. External bleeding was minimal at the time of injury and a massive subcutaneous hematoma did not develop. The left arm became paralyzed at the moment of injury and did not exhibit evidence of improvement for two months. Return of function was marked first by flexion of the forearm, and then by extension of the wrist. Sensory loss was noted over the entire arm after injury, but regressed slowly until it involved the outer aspect of the forearm, the thumb, and first three fingers. After hospitalization in the Zone of the Interior, an aneurysm of the first part of the axillary artery was disclosed, and he was referred to the Ashford General Hospital for treatment.

Examination noted a small, well healed wound of entry 4 cm. below the outer third of the left clavicle and a larger, irregular scar denoting the wound of exit lateral

to the tip of the left scapula. In the left infraclavicular space an oval, expansile mass, 3 x 2 cm. was palpable. Upon auscultation, a faint, systolic bruit was audible, which radiated into the distal third of the extremity. Blood pressure in the right arm was 122/78, in the left 110/76. The radial pulse on the left was weaker than on the right; the ulnar pulse was absent.

Neurologic study noted complete involvement of the musculocutaneous nerve, an incomplete lesion of the median nerve and minimal disturbance of the radial nerve, with corresponding muscle dysfunction. There was an area of hypalgesia and anesthesia corresponding to the sensory distributions of the lateral antebrachial cutaneous and median nerves. On August 16, 1943, a false aneurysm of the first part of the left axillary artery was resected. At operation, an extensive gap in the musculocutaneous

FIG. 5.—Case 5: Note operative approach to aneurysm of axillary artery and neural defects.

nerve was observed. The mass of the aneurysm compressed the median nerve in particular. There was no demonstrable change in the vascular supply to the extremity following operation. Beginning improvement in the neural defect is detectable at this time, three months after operation (Fig. 5).

Case 6.—*J. K. C., Hosp. No. 4246: Rifle wound of right chest with subsequent shrapnel wound of right shoulder on April 23, 1943. Early paralysis of right arm with rapid functional recovery. Residual serratus anterior and triceps muscles paresis. Arterial aneurysm of lateral thoracic artery resected.*

This soldier sustained a rifle bullet wound of the upper right chest on April 23, 1943. He remained on the field under fire until the following day when he was again injured by a shrapnel fragment in the right shoulder. At this time, the right arm was paralyzed with the hand and fingers in flexion spasm. The hand and arm recovered normal function, as far as the patient could tell, in the following two months. Chest pain persisted, and he was evacuated to the Zone of Interior.

Upon admission, October 25, 1943, a rounded, pulsating mass was palpated high in the right axilla in the course of a routine examination. It appeared to be separate

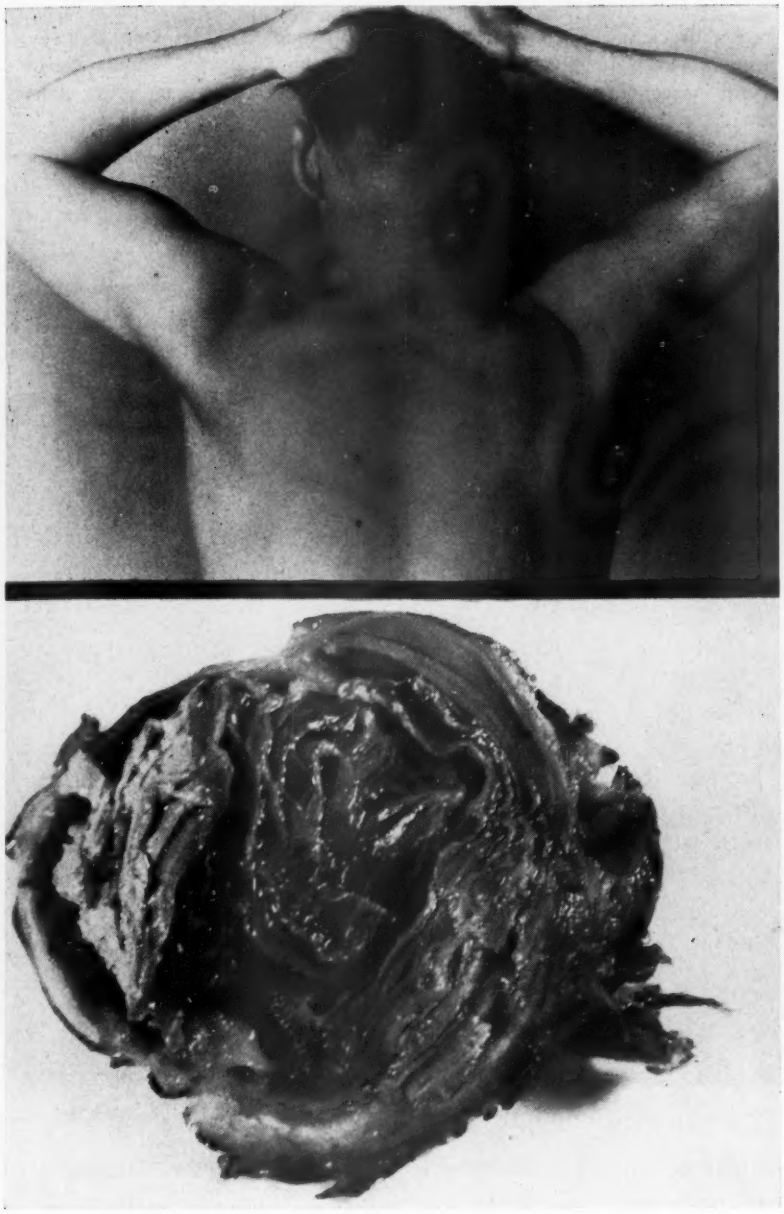

FIG. 6.—Case 6: Note triceps atrophy. Below, bisected aneurysm of lateral thoracic artery, showing lamination of clot.

from the pulsation and course of the axillary artery. A systolic nontransmitted bruit was present over the mass and both pulsation and mass could be obliterated by subclavian artery compression. A small scar was present on the anterior aspect of the right shoulder. Neurologic examination disclosed atrophy of the triceps muscle with associated weakness of extension of the upper arm. The muscle mass of the right deltoid was diminished on the right and early abduction movement of the right arm was weak. There was moderate flaring of the vertebral tip of the right scapula.

On November 2, 1943, exploration of the right axilla disclosed an arterial aneurysm of the long thoracic artery measuring 4 cm. in diameter. The long thoracic nerve curved around and was imbedded in the wall of the aneurysm. After resection of the aneurysm inspection of the axillary and radial nerves revealed no gross abnormality (Fig. 6).

In Makins' series, nerve injuries occurred in ten of 43 instances of injury to the brachial artery. Of striking interest is the fact that only one arteriovenous aneurysm was found in Makins' patients as compared with 12 of the arterial variety. One A-V aneurysm of the brachial artery with associated nerve involvement was encountered in this series. Due to associated infection, it has not as yet been treated. A brief description of this patient is noted under Case 12.

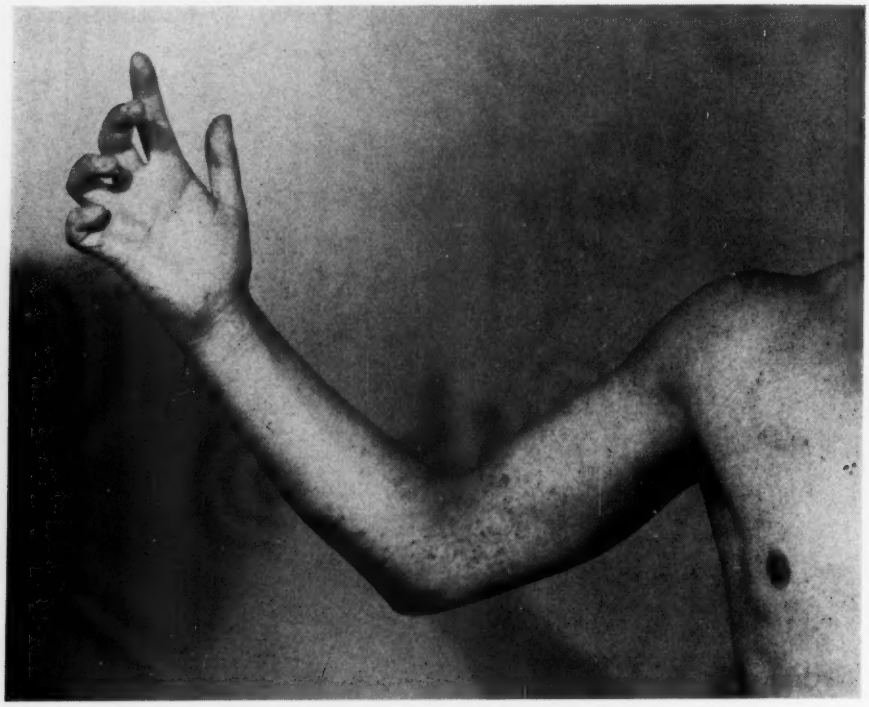

FIG. 7.—Case 7: Postoperative appearance following endo-aneurysmorrhaphy of brachial artery. Note obvious ulnar nerve deformity.

Two arterial aneurysms of the brachial artery in the antecubital space associated with nerve injury were observed. Both demonstrate the value of resection of the aneurysm as compared to endo-aneurysmorrhaphy when concomitant nerve injury is present.

Case 7.—*E. W. B., Hosp. No. 1403: Gunshot wound of right upper forearm. Immediate numbness of ulnar aspect of hand with weakness of intrinsic hand musculature. Obliterative endo-aneurysmorrhaphy of brachial artery with second-stage extraneural neurolysis of ulnar nerve.*

This soldier was shot by a rifle bullet on March 23, 1943. The missile entered the lateral aspect of the middle third of the right forearm, traversed the arm diagonally and made its exit slightly above the medial condyle of the humerus. The wound of exit

bled profusely and a tourniquet was applied. He noticed immediately weakness of the hand grip and loss of feeling along the inner aspect of the forearm, the ulnar aspect of the hand, and the fourth and fifth fingers. There was a transient infection in the wound of exit.

Examination of the right upper extremity, May 7, 1943, showed a tubular swelling of the proximal half of the forearm with a fluctuant area 4 cm. below the medial condyle of the humerus. There was a systolic pulsation together with a systolic bruit over the fluctuant area. Elsewhere the skin and subcutaneous tissues were firm and indurated without local heat or change in the overlying skin. Pressure in the region of the ulnar nerve adjacent to the wound of exit caused a tingling paresthesia. There was loss of function in the flexor carpi ulnaris and the two medial heads of the flexor digitorum profundus and the intrinsic musculature of the hand innervated by the ulnar nerve. The little finger and the ulnar aspect of the hand were analgesic and anesthetic. There was a narrow band of hypalgesia along the lateral margin of the lower half of the forearm. Obliterative endo-aneurysmorrhaphy of the brachial artery was done on May 17, 1943, with no alteration in the peripheral vascular supply to the hand. On July 14, 1943, the ulnar nerve was found divided in the thick scar tissue at the site of the aneurysmal sac. An end-to-end anastomosis was carried out with tantalum wire. No return of function is evident at the end of four months (Fig. 7).

Case 8.—*H. A. B., Hosp. No. 3009: Gunshot wound of left forearm. Immediate numbness and weakness of hand. Arterial aneurysm of brachial artery in antecubital space. One-stage resection of aneurysm and neurolysis of ulnar and medial nerves. Slow improvement in neurologic status.*

This patient was shot by a .30 caliber rifle bullet on July 3, 1943. The missile entered the left forearm on its anterior and lateral aspect and emerged just above and posterior to the medial epicondyle of the humerus. He noticed immediate numbness and weakness of the hand. There was marked swelling of the elbow, forearm, and hand within two hours of the injury but there was no external hemorrhage. On the 17th day following injury, an incision was made below the antecubital space and much blood clot was evacuated. On admission, August 16, 1943, slight motion of the wrist and of the third finger had appeared. There had been no improvement in the original sensory loss.

Healed wounds of entry and exit were noted corresponding to the path of the missile. The left upper forearm was swollen and indurated. An oval mass, 6 cm. in length, was palpable on the medial aspect of the forearm below the antecubital fold. There was an expansile pulsation in the mass, synchronous with cardiac systole. A loud systolic bruit was audible upon auscultation. Compression of the brachial artery in the arm caused the mass to become smaller and the bruit to disappear. The ulnar pulse was absent. The radial pulse was weaker on the left side than on the right.

Motor examination of this extremity showed disuse atrophy of the arm musculature. Minimal radial extension of the wrist was possible. Slight extension of the third finger at the metacarpophalangeal articulation was elicited. No other movements of the hand musculature were possible. There was marked contracture of the flexor tendons to all fingers. The entire hand showed cutaneous atrophy and loss of sweating. Analgesia and anesthesia were present over the sensory distributions of the ulnar and median nerves in the hand.

On September 7, 1943, the arterial aneurysm was resected. The median nerve was compressed by the mass of the aneurysmal sac and the ulnar nerve was buried in a mass of scarred, indurated muscle tissue medial to the aneurysm. It was observed that widespread muscle damage was present in the bellies of the flexor musculature of forearm. Slight improvement in the neurologic status has been evident since operation (Fig. 8).

The peripheral nerve injuries visualized at operation in the preceding four patients differed grossly in but two respects from peripheral nerve injuries observed in the absence of vascular injuries. In the first place, to

FIG. 8.—Case 8: Arterial aneurysm of brachial artery with ulnar and median nerve paralysis. Preoperative photograph. Note drainage scar. (Insert) Operative specimen.

the usual pathologic findings of complete or partial nerve section, contusion or compression by scar tissue, is added the factor of compression by the mass of the aneurysmal sac. It is noteworthy in these cases, and in the few recorded in detail in the earlier literature, that the neurologic defect usually appears full blown at the moment of injury. Spontaneous return of neurologic function rarely appears and operative repair of the neural pathology is mandatory. In the second place, the presence of a vascular lesion, be it arterial transection or aneurysm with concomitant nerve injury, tends to exaggerate and make permanent the existing neural dysfunction through local and general alteration in the nutrition of muscle fibers. The frozen, contracted hand, resulting from acute arterial occlusion is well recognized, and if this deleterious factor be added to a neural defect, the resulting deformity may be irremediable.

It is of interest that in Case 7 the question of abscess formation rather

than arterial aneurysm was considered, and in Case 8 actual incision was done. In Fromme's Cases 1 and 25, similar supposed abscesses were incised with resultant severe hemorrhages, and in Case 46, identical with the last two of this series, the diagnosis of abscess was entertained briefly.

Finally, it may be stated, unequivocally, that a single, inclusive surgical approach to the vascular and the neural lesions is preferable to surgical repair of either lesion at separate operations.

III—VESSEL AND NERVE INJURIES OF THE LOWER EXTREMITY

Peripheral nerve injuries occur with arterial or arteriovenous aneurysms of the lower extremity but with less frequency than with those of the upper extremity and neck. They may involve the sciatic nerve in traversing wounds of the thigh, and the peroneal and tibial components in large, open wounds of the popliteal space. In only five of 85 instances of popliteal artery injury were neural structures involved in Makins' series, and the incidence in other major reports is correspondingly low.

Case 9.—*J. C. B., Hosp. No. 2109: Machine gun wound of left femoral region. Immediate paralysis and loss of sensation in extremity with rapid return of function. Resection of A-V aneurysm of common and deep femoral artery and vein.*

This officer was struck in the left femoral region with a machine gun bullet on April 29, 1943. A perforating wound of the thigh was sustained with the wound of exit in the left buttock. This extremity was paralyzed and made anesthetic at the time of impact, but motor and sensory defects cleared within a few hours. A profuse external hemorrhage occurred, which was controlled by pressure. There was no evidence of circulatory deficiency, but pain in the calf and popliteal space developed early. This distress increased with the appearance of a harsh bruit over the femoral triangle, of dilated leg veins and some peripheral cyanosis. On June 11, 1943, the left femoral artery and vein were ligated proximal to the fistula without alteration in the peripheral circulation and without effect upon the aneurysm. At this time, cardiac studies were normal.

Upon admission, July 11, 1943, examination showed induration in the left femoral triangle. About 8 cm. below Poupart's ligament was a small wound of entry beneath and lateral to a well-healed surgical incision. A second surgical incision was situated lateral to the first. The scar representing the wound of exit was noted in the left buttock. Beneath the larger incision a loud, harsh bruit was audible throughout systole and diastole, accentuated during the former phase. Occlusion of the femoral artery above the fistula diminished but did not cause the bruit to disappear. Such occlusion caused a transitory slowing of the pulse rate. The peripheral pulses were absent in this extremity and there was slight, dependent cyanosis of the foot. There was definite enlargement of the heart to the left, confirmed by fluoroscopy. Neurologic examination demonstrated only a diminution of the left knee kick and weakness in dorsiflexion of the left great toe. There were no sensory changes. The left sciatic nerve was somewhat tender and pressure over it increased the peripheral pain.

On August 4, 1943, quadruple ligation and resection of the arteriovenous fistula was performed. His convalescence was uneventful and the radiating pain slowly diminished.

Case 10.—*M. J. R., Hosp. No. 2957: Multiple injuries of trunk and extremities from land mine, including extensive laceration of right popliteal space. Development of popliteal A-V aneurysm with involvement of lateral cutaneous sural nerve. Quadruple ligation and excision of aneurysm, neurolysis.*

This officer in the Medical Corps received multiple injuries of the trunk, abdomen, upper and lower extremities from the explosion of a land mine in April, 1943. He re-

mained dazed and disoriented for several days as the result of blast concussion. At this time he recognized the presence of a right ulnar nerve paralysis and appreciated an area of numbness over the lateral aspect of the right lower leg. He stated that there was a profuse hemorrhage from a broad laceration of the right popliteal space at the time of injury, necessitating several plasma and whole blood transfusions. One month following the injury, the ulnar nerve paralysis began to recede. At this time, he became conscious of a continuous thrill in the popliteal space and was the first to recognize the presence of arteriovenous aneurysm of the popliteal vessels. He noticed no symptoms referable to cardiac strain except shortness of breath, and he did not feel that the peripheral blood supply to the involved extremity was impaired.

FIG. 9.—Case 10: Multiple injuries from explosion of land mine. Postoperative photograph, following excision of popliteal A-V. fistula.

Examination was noteworthy for the presence of 28 separate scars over the posterior surface of the trunk and lower extremities, characteristic of multiple injuries from the explosion of an antipersonnel land mine (Fig. 9). Over the inferior aspect of the right popliteal space was a broad, irregular scar, extending laterally beneath the tibial tubercle. Above this scar and rather high in the popliteal space the tissues appeared full. Upon palpation at this point, a strong continuous thrill could be felt, and upon auscultation, the bruit was continuous but accentuated with systole. There was no evidence of cardiac damage and the peripheral blood supply of the foot was normal. Hypalgesia and hypesthesia were present over the sensory distribution of the lateral cutaneous sural nerve. There was no evidence of motor dysfunction in this extremity. Neurologic study elsewhere disclosed only hypalgesia over the cutaneous area innervated by the ulnar branch of the medial antibrachial cutaneous nerve. On August 24, 1943, the arteriovenous aneurysm was excised after quadruple ligation. At operation, the peroneal nerve was compressed by scar tissue and an external neurolysis was done. The neurologic defect has improved steadily and he has returned to duty.

Case 11.—*P. W. P., Hosp. No. 3008: Shrapnel wounds of right popliteal space May 5, 1943. Profuse hemorrhage, leg pain and diminished peripheral circulation. Lumbar sympathectomy with improvement in circulation. Resection of popliteal A-V aneurysm. Normal neurologic examination.*

This soldier received wounds of the right knee, leg and left shoulder from fragments of an 88 mm. shell on May 5, 1943. Profuse hemorrhage occurred from a wound in the right popliteal space which was débrided and the hemorrhage controlled. No injury to major vascular or neural structures was reported. The right lower leg and foot remained cold and cyanotic, with considerable aching pain. On July 17, 1943, a right lumbar sympathetic ganglionectomy was carried out with partial relief of pain, and considerable improvement in the peripheral circulation.

Upon admission, August 29, 1943, there was a flat, healed scar in the right popliteal space and a second operative scar in the posterior upper third of the calf. A shrill bruit was audible over the popliteal space, extending throughout systole and diastole and accentuated during the former. Occlusion of the fistula did not change the pulse rate. The right posterior tibial pulse was absent, but the right foot was warmer than the left. There were no abnormal cardiac findings. Neurologic examination was normal (Fig. 10).

On September 6, 1943, quadruple ligation and excision of the arteriovenous aneurysm was done. There was no change in the peripheral circulation and his convalescence was uneventful. The tibial and peroneal nerves, although closely adherent to the fistulous mass, appeared normal at operation.

FIG. 10.—Case 11: Preoperative photograph of popliteal arterio-venous aneurysm.

Case 12.—*S. M., Hosp. No. 3528: Multiple wounds of right arm, chest and left leg on July 11, 1943. A-V aneurysms of left femoral and right brachial vessels. Minimal hypalgesia in left popliteal space. Quadruple ligation and an excision of femoral A-V fistula with aneurysmal sac on September 7, 1943.*

This patient, in brief review, developed an arteriovenous aneurysm of the right brachial artery, upper third, and a comminuted compound fracture of the right elbow, with associated nerve injuries and an A-V aneurysm of the left femoral vessels.

Upon admission to this hospital, a pulsating, expansile mass was noted on the superior and medial aspect of the left thigh. There was a palpable thrill and audible bruit lasting throughout systole and diastole, with systolic accentuation. Occlusion of the fistula did not alter the pulse rate. The peripheral circulation was adequate and there was no evidence of cardiac damage except a constant elevation of the pulse rate (110 per min.). Neurologic examination showed, in this extremity, only hypalgesia and hypesthesia in the popliteal space, suggesting involvement of some of the fibers of the posterior femoral cutaneous nerve. Quadruple ligation and resection of the fistula and adjacent aneurysmal sac were carried out on September 7, 1943. His convalescence was uneventful from this localized disturbance. Operation upon the brachial aneurysm has not yet been carried out because of local infection in the associated compound fracture (Fig. 11).

SUMMARY

It appears probable that more vascular and nerve injuries of various types will be seen in this war than in World War I, because of improved methods for the control of shock and infection and because of the increased incidence of multiple injuries.

The diagnosis and treatment of aneurysms and peripheral nerve trauma have not been discussed in this paper. However, it should be pointed out that certain basic principles in the treatment of aneurysms must be observed or else failure is sure to follow. For example, the treatment of aneurysm, whether it be arterial or arteriovenous, is never a matter of emergency unless it is rapidly progressing in size or has ruptured, or unless heart failure is impending. Time should be allowed for collateral circulation to develop, usually a matter of three or four months. The use of artificial means to develop collateral circulation, usually proximal compression of the vessels, should be employed. Moreover, the eradication of an arteriovenous fistula

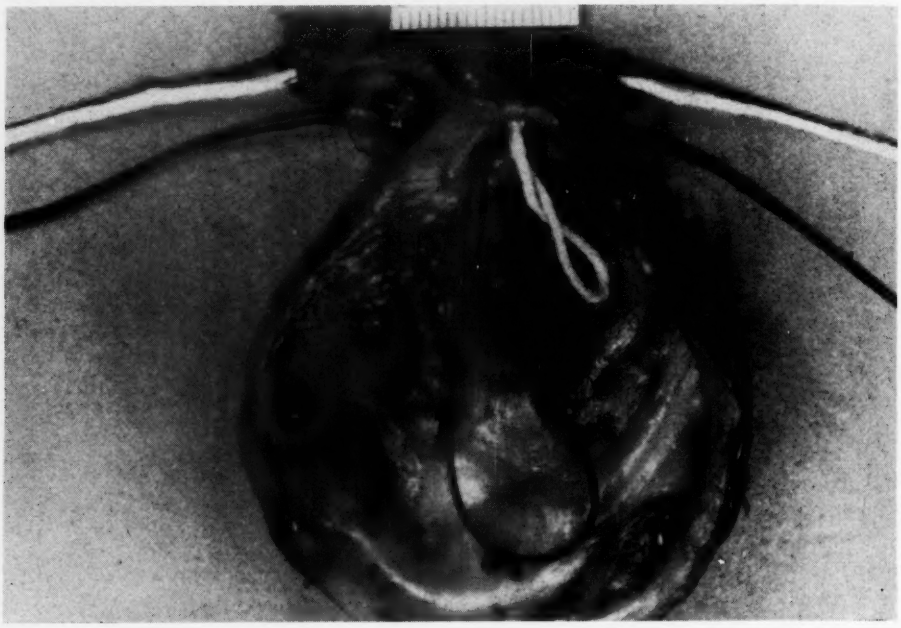

FIG. 11.—Case 12: A-V. fistula with communicating arterial sac. Note white string in artery and black string in vein.

is accomplished only by quadruple ligation and excision. Any other method usually results in recurrence. Partial measures, such as proximal ligation of the artery, may lead to gangrene of an extremity. Complete excision of an arterial aneurysm, together with nerve repair, as a single operative procedure, is the method of choice. When major vessels are involved and where the nutrition to the extremity is questionable, the endo-aneurysmorrhaphy of Matas is preferable. This applies particularly to the common femoral and other major arteries.

It is well recognized in all peripheral nerve surgery that a conservative attitude should be taken regarding technical procedures. This is particularly important in injuries associated with blood vessel trauma because of the added factor of compression by an aneurysm. This factor, producing added nerve damage in the false aneurysm, is not often present in arteriovenous fistulae, which are rarely large enough to produce pressure damage. Nerve injury

must occur at the time of formation of the fistula, through the same inflicting force, and may be due to concussion, contusion, or to actual section of nervous tissue.

In many instances, at the time of the original injury, there is evidence of extensive nerve damage. By the time that vascular repair is indicated, the neural damage may have resolved itself to a large extent, leaving a residual neurologic defect, which may be repaired at the time of operation upon the blood vessel.

Recognizing the slowness with which nerve regeneration takes place and desirous not only of attaining the best functional result, but also of making studies for future reference and treatment, the Surgeon General has adopted a policy of treating all peripheral nerve lesions until maximum hospital benefit has been obtained. Even where soldiers are returned to duty, they are ordered back to the hospital where last treated for periods of observation every three months.

Finally, it is emphasized that the traumatic syndrome of Vernet and Villaret, *viz.*, involvement of the last four cranial nerves, is often associated with blood vessel damage in the neck, due to the close proximity of these tissues.

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CONTROL OF PAIN IN POSTTRAUMATIC AND OTHER VASCULAR DISTURBANCES*

THE RÔLE OF THE SYMPATHETIC NERVOUS SYSTEM IN THE TREATMENT OF PERIPHERAL VASCULAR DISEASES

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THE RÔLE of the sympathetic nervous system in the treatment of vascular diseases is very controversial. Much knowledge concerning the functions of the sympathetic nervous system has been acquired from clinical trial which initially deserved only the title of clinical experimentation. Perhaps this appearance of much of Leriche's work led to incredulence and delayed acceptance. One of the most controversial of all phases of the sympathetic nervous system, is the part it plays in pain, especially deep pains of the vessels, bones and muscles. No animal experimentation has advanced knowledge so well as clinical experiments in this field. No absolute conclusion is justifiable regarding such pain and the sympathetic nervous system, but it is surprising what may happen with regard to certain pains when the sympathetic nerves are interrupted.

MATERIAL

On my private services at the Touro Infirmary and Baptist Hospital, 62 patients have been admitted whose condition demanded temporary or permanent interruption of the sympathetic nervous system. These cases have given opportunities for much clinical investigation. The diagnoses in the different cases were as follows:

Raynaud's disease	4
Posttraumatic syndrome (pain, edema, causalgia, Sudeck's atrophy)	19
Thrombo-angiitis obliterans	14
Thrombophlebitis or postthrombophlebitic edema	20
Arteriosclerosis	3
Hyperhidrosis unilateralis	1
Herpes zoster	1
	62

Twenty-two operations were performed to permanently interrupt the sympathetic nervous system. Eleven of these were lumbar ganglionectomies and 11 were preganglionic sections of the dorsocervical sympathetic nerve trunk for the upper extremities. The dorsocervical operation was performed seven times for the posttraumatic syndrome and four times in two patients for Raynaud's disease. Lumbar ganglionectomy was performed nine times (in eight patients) for thrombo-angiitis obliterans and twice for postthrombophlebitic edema.

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The pains of vascular diseases are varied in cause and type. In many vascular diseases, pain is the predominant, sometimes even the only disabling symptom. Measures which relieve the pain even though they may not return the vascular system to normal, may rehabilitate the patient to an extent which permits a useful if not a very active existence. Too frequently, the diagnosis of an arterial vascular disease excites predominant thoughts of impending gangrene and its prevention, when in reality, lesser degrees of arterial obstruction or spasm not converging on a state of gangrene, may produce very severe painful symptoms which disable to the extent that the patient is a liability instead of an asset to himself and his family. With pain, a man with a severe arterial impairment, occlusive or vasospastic, may restrict his activities and live a very plausible existence, but with constant pain, he becomes totally disabled. Most of the pains in vascular diseases are diffuse, deep, often intense, and there are some reasons to believe that the sympathetic nervous system plays a large part directly or indirectly in the transmission of such pain.

Edema, a cold clammy hand or foot, and "poor circulation" associated with pain or soreness, may persist for a surprisingly long time after an injury remaining as the only vestige of the injury to mar what might be judged otherwise a perfect result. The vascular changes occurring after trauma are not fully appreciated. Fractures and major soft tissue injuries, and even certain minor injuries, may be followed not only by the immediate changes in the vascular system, but also by vascular changes which are chronic and persistent. These changes are vasodilatation or vasoconstriction or both. Whether the pain or vasospasm is antecedent or whether they are concomitant and not cause and effect, has not been determined; nor is the nature of this pain well understood. The coldness and "poor circulation" may be subclinical, only detectible by a precision instrument such as a thermocouple (dermotherm). Joint stiffness may linger not because of ankylosis, but because edema and swelling around the capsule and pain on attempted motion is so severe, a residual which could be quickly obviated without the pain, is prolonged because of it. This pain of joints is similar in some respects to the other pains of causalgia and Sudeck's atrophy.

The pain of other vascular diseases simulates in many instances that of the posttraumatic syndrome. The severe rest pain of thrombo-angiitis obliterans is explainable not only by ischemia. If so it would be at first most peripheral and advance upward as the ischemia progresses. Instead it often begins in the middle of the foot. Spasm of the vessel itself and not the resulting ischemia may be responsible for pain. The kinetic pain or claudication may be entirely different. The pain of Raynaud's disease is most intense, the most disabling part of the disease. Its relief seems to be proportionate to the relief of vasospasm. When we have been taught to think the conveyance of pain, all pain, is the property of somatic nerves, it is too violent a change to have to accept easily, the possibility that another type of nerve namely the sympathetic system, may convey severe pain.

Leriche maintains the pain is conveyed by the sympathetic nerves. Davis and Pollock think it is carried over the somatic nerve, but that the irritated sympathetic nerves cause a metabolic product which excites pain sensation in the sensory nerves. Miller and de Takats, after careful plethysmograph studies, concluded that vasodilatation occurs in some cases and vasoconstriction occurs in others. They conjectured that vasoconstriction may occur in the finer vessels while vasodilatation is occurring in the larger.

It is difficult to classify clinically sympathetic nerve system pains. There are at least four or five groups: (1) Posttraumatic pain. (2) Causalgia. (3) Sudeck's atrophy. (4) Sympathalgia. (5) Nontraumatic reflex vasospasm (from inflammation, thrombosis, embolus, *etc.*).

Under this classification, posttraumatic pain is a condition in which the objective findings are minimal with subclinical vasospasm and possibly edema. Causalgia portends pain with more objective findings, edema, paresis of a nerve and sometimes pain only in the distribution of a nerve. In Sudeck's atrophy, pain is associated with an osteoporosis. Sympathalgia is sympathetic nerve pain not necessarily associated with traumatic onset. Another group of painful conditions associated with vasospasm but not caused by external trauma are those of inflammation, embolus, thrombosis, and similar causes.

Whatsoever be the phenomena associated with posttraumatic pain, it is true that interruption of the sympathetic pathways by infiltration with novocain, or by surgical section, usually dramatically relieves the patient. Moreover the pains of other vascular diseases may be relieved by sympathetic interruption. What relief of pain is afforded purely by the relief of vasospasm is debatable. It is feasible to shorten the convalescence of patients with persistent pain following injury by repeated paravertebral sympathetic nerve blocks or if warranted by surgical section of the sympathetic nerves. By sympathetic nerve block is meant infiltrating the region of the sympathetic ganglia with novocain, *i.e.*, in the region of the stellate and the second and third dorsal ganglia for the upper extremity or through four needles placed adjacent to the vertebral bodies in the 1st, 2nd, 3rd, and 4th, lumbar interspaces for the lower extremity. A few examples will explain better what is meant by such therapy:

(1) A man, age 41, came under observation 19 weeks after an accident in which he sustained a supracondylar fracture of the left forearm. Even immediately after the plaster encasement was applied, he had severe persisting pain in the arm and hand. The encasement had been removed six weeks after the accident, and his arm and hand remained stiff and painful. Examination revealed a condition which resembled the early picture of Volkmann's ischemic contracture, a stiff hand, wrist, fingers and elbow. The elbow motion was not more than 20°. The motion of the fingers and wrist not more than 10°. Moreover there was edema of the forearm and hand, atrophy of the muscles, and extreme pain on the slightest motion of the joints. He had received physiotherapy elsewhere, but the intense pain prevented any active mobilization of the joints.

Repeated paravertebral sympathetic nerve blocks with novocain were done. Each time one was done, in addition to the Horner's syndrome, the hand became warm and there was a dramatic relief of pain even on forced motion of the joints. This relief of pain lasted all of one day and it returned to a lesser extent on the following day. The joints were quickly mobilized and after three weeks he was dismissed from the hospital with comparatively little limitation of motion in the joints. Eventually function returned so completely to the muscles and joints, that the result could be regarded as practically perfect.

In this instance operation to section the sympathetic nerve may have been justifiable, but the result could not have been better following operation. This also illustrates the value of repeated temporary sympathetic nerve interruption in patients with early Volkmann's ischemic contracture.

(2) A carpenter, age 38, caught the terminal phalanges of the index and middle finger of his left hand in a machine. Compound fractures of the terminal phalanges resulted and both fingers were badly contused and lacerated. The end of the third finger was almost amputated. This was sewed on and in due time the wounds healed and union of bone resulted. Even after eight weeks, however, the severely scarred fingers were so sensitive he could not return to work. (Plate I, b1 and b2.) They were hyperesthetic to the extent that he could not hold a nail and even putting his hand in his pocket caused so much pain in the hypersensitive fingers and tender scar, that he refrained from doing it. He was emotionally stable and by no means was he a malingerer. At this phase of his convalescence, pain alone was obstructing his return to work. Then (at the end of eight weeks) the dorsocervical sympathetic chain was blocked with novocain by the Leriche method. With the patient recumbent, a pillow under the shoulders and the head slightly hyperextended and rotated to the opposite side, a wheal was made one finger's breadth above the junction of the inner and middle third of the clavicle. A No. 24-gauge spinal needle was inserted through the skin wheal and in the coronal plane, and at a 45° divergence from the sagittal plane, so that the needle went backward and inward slowly until it struck bone—the lateral surface of the body of the first dorsal vertebrae. After aspirating and obtaining nothing through the syringe, five cubic centimeters of one per cent novocain was injected. Then the point of the needle was withdrawn and in the same oblique plane, the butt of the needle was deviated cephalad in order to throw the point farther down onto the body of the 2nd dorsal vertebra as it was reinserted. The needle was changed in the coronal plane several times to strike the first or second rib and infiltrate the gutter between the ribs and the lateral surfaces of the first two vertebra. Ten cubic centimeters of novocain was delivered. A Horner's syndrome developed within two or three minutes and within five to ten minutes, the hand of that side was warmer even, and drier, than the opposite hand. The hyperesthesia and tenderness of the fingers dramatically vanished and it was possible to press the injured finger tips where 15 minutes before, they were so sensitive, he could not stand the lightest touch. The

scarred area remained less sensitive all that day. The next day the pain returned, but to a far less extent. His finger remained much better than it had been. One week following the first sympathetic nerve block, another block was performed. This time the hyperesthesia disappeared entirely. It returned to a minor extent only, so that he could handle nails and do his work. He returned to work a few days thereafter, and his disability was shortened by weeks.

(3) This case is an example of one in which preganglionic sympathectomy was judged advisable for relief of causalgia, a pain following a previous operation. A woman, age 29, had severe pain in her left arm, which was more pronounced in the region of a scar on the anteromesial aspect of the forearm. (Plate I C1 and C2). Two years ago, in another city, a small mass was removed from the forearm. It recurred and after a few months it was removed again. She was told it was a neurofibroma. Ever since then, the scar has been extremely sensitive and pain radiates up and down the arm. The scar was very tender and pressure on it caused a pain to shoot up and down the ulna side of the arm. Examination failed to reveal any mass in the arm. A roentgenogram showed a rudimentary cervical rib. Considerations in the diagnosis included, scalenus anticus syndrome, recurrent neurofibroma, and causalgia. A sympathetic nerve block was performed. A Horner's syndrome developed, and the pain was completely relieved and tenderness of the scar disappeared, but, all symptoms returned with severity the next day.

Several days later, the following operation was performed: The area under the scar was explored; no mass was found. A small amount of scar was removed. The ulna nerve was found to be normal. Because these findings were expected, the patient had been draped so that a dorsocervical preganglionic sympathectomy could be done immediately. Through an anterior approach the scalenus anticus muscle was severed. The dorsal sympathetic chain was severed below the third dorsal ganglion and the rami communicantes to the second and third dorsal ganglia were severed. The proximal end of the nerve trunk was sutured to the scalenus medius. The rami communicantes to the stellate ganglion were left intact. The patient was completely and permanently relieved of her pain. The left hand and arm remained warm and she did not develop a Horner's syndrome because the rami communicantes to the stellate ganglion were not disturbed.

In these and similar cases interruption of the sympathetic nerve system by repeated novocain blocks or by surgical section may prove of tremendous value in restoring the patient to normal. The question as to how often to do the novocain blocks depends on the case. It is rare that one block will give permanent relief for a condition which justifies its being done at all. As a tentative routine plan, I prefer to block the nerve every other day

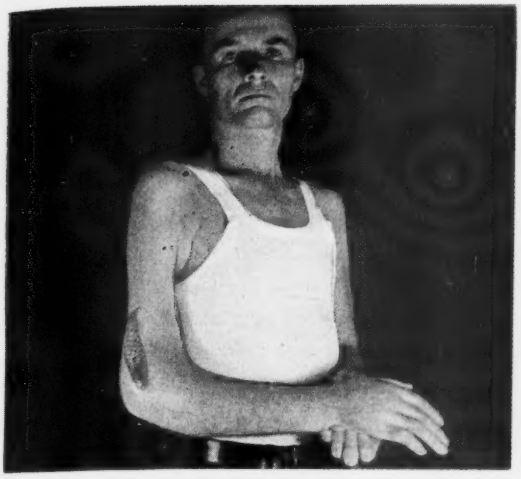
PLATE I

A1 Man age 30. Three months prior to this his arm was caught in a conveyor belt. When first seen he had edema and pain in arm with markedly stiff and painful joints, A2 after repeated paravertebral sympathetic nerve blocks which helped to clear the edema and permitted mobilization of the joints. End result not perfect, but good enough to permit him to return to his former job "pushing long iron rods" in a container factory.

B1 and B2 injury to index and middle fingers left him with hyperesthesia and extremely sensitive scars. Paravertebral sympathetic nerve blocks promptly relieved the pain and paresthesia. (See text).

C1 and C2 A woman age 29 with causalgia of left arm. Exploration of forearm through old scar revealed no local cause. Preganglionic dorsocervical sympathectomy resulted in relief of pain.

PLATE I



A1

A2

B1

B2

C1

C2

for three times, then if necessary, every third day for an additional time or two. As for alcohol, the danger of neuritis precludes its use in the dorsocervical region. Even in the lumbar region it may produce neuritis. Moreover, alcoholic injection is inefficient. True, it may produce some prolonged vasodilatation, but the effect is less marked than that immediately following novocain infiltration and is certainly less marked than following surgical extirpation. Either repeated sympathetic novocain blocks accomplish the desired result, or, if more permanent, more complete vasodilatation is desired, operation is indicated. Sympathectomy is a relatively safe operation, as safe as appendicectomy, and its recovery period is equally short.

It is difficult to clearly designate the indications for preganglionic section of the sympathetic nervous system for the relief of pain. Such varieties of conditions are involved that it obviously is a problem to be individually solved for the patient in question. In general, however, operation is not indicated in instances where a few repeated sympathetic nerve blocks accomplish the desired result, and operation is never indicated for relief of a pain which sympathetic nerve block does not temporarily mitigate or completely abolish. The operative approach for preganglionic section of the dorsocervical sympathetic nerves (third and second dorsal and stellate ganglia) may be from behind (Smithwick) or the front. Though each has its advantages (perhaps the posterior approach affords the most room), I have used the anterior approach more frequently. This is the operation described by Telford, in 1935. The sympathetic nerve trunk is severed below the third dorsal ganglion. The rami communicantes to the second and third ganglia are severed. I sew the proximal end of the nerve trunk to the scalenus medius muscle. It is my experience that in the posttraumatic syndrome preganglionic sympathetic nerve section is followed by a persisting warm and dry extremity but not so in Raynaud's disease where vasospasm is more apt to recur. The uniformity of persisting vasodilatation following sympathectomy for causalgia and the uncertainty of its duration following the same procedure in Raynaud's disease is evidence against Raynaud's disease being a fault of the sympathetic nerve trunk.

After preganglionic section of the dorsocervical sympathetics for causalgia, the upper extremity remains warm and dry indefinitely. The vasodilatation measured by the thermocouple is a little less marked after seven days than it is on the first day, but it still remains warmer than the opposite extremity. In Raynaud's disease the result is not similar. In the latter disease, preganglionic section of the sympathetics may be followed by a recurrence of the vasospasm—in ten days, two weeks, six weeks, and the time of its occurrence may be determined largely by the first exposure of the patient to cold. It is not regeneration of nerves which makes the difference. It is difficult to believe that regeneration occurs in Raynaud's disease only, and not after, preganglionic nerve section for causalgia. The fact that an operation which will cause permanent vasodilatation in a person who is relatively normal and is followed by a recurrence of vasoconstriction of finer terminal arterioles and capillaries in Raynaud's disease, is evidence in favor of the belief that

the cause does not act through the sympathetic nerve outflow, but by a local or circulating factor which overcomes the tendency to vasodilatation following sympathectomy. It is my experience that the vasodilatation following preganglionic section for Raynaud's disease persists in the skin of the arm but diminishes in the extreme part of the upper extremity, *i.e.*, the fingers. Moreover, the larger arteries pulsate well and fully and are not under the influence of vasospasm, whereas, the extremely fine arteries of the fingers are under the most

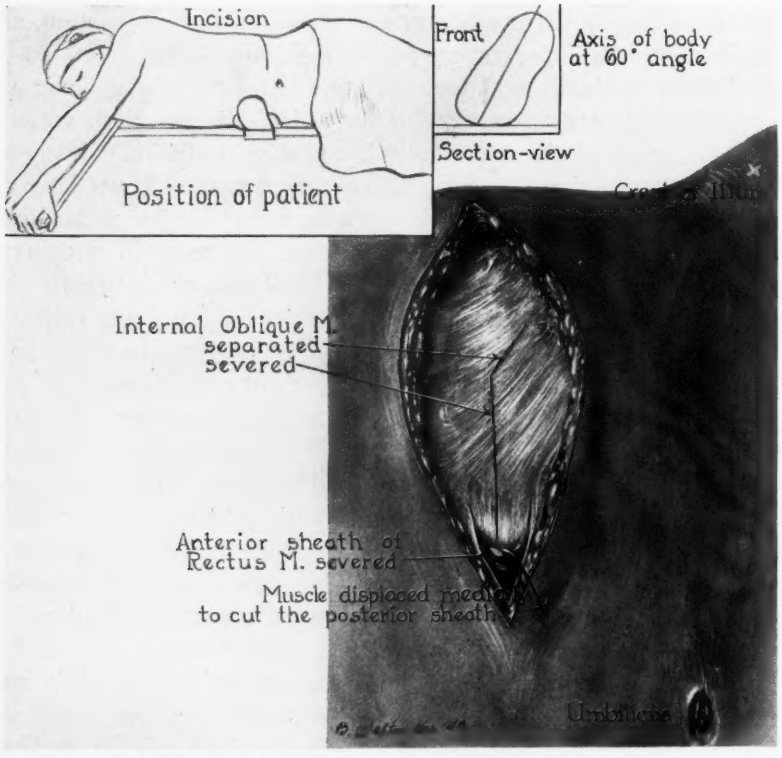


FIG. 1.—Lumbar sympathetic ganglionectomy. Patient in lateral position and axis of body tilted backwards. Needles are placed posteriorly at the first lumbar interspace and at the fourth lumbar interspace just as for paravertebral sympathetic nerve block. These serve as landmarks for the second to fourth lumbar ganglia. Incision is made from the eleventh rib forward to just above the umbilicus. The aponeurosis and fibers of the external oblique are severed in the line of incision.

intense vasospasm and the fingers are very painful. Moreover, sudomotor function after the same type of sympathectomy remains paralyzed which is evidence indicating that it is not the type of operation which fails, but that it is the type of disease which makes it fail. Raynaud's disease appears not to be fundamental disturbance of the sympathetic nervous system. Lewis, following careful studies, believed the condition to be a local fault. The release of the normal sympathetic nerve function does cause a temporary vasodilatation, but it does not attack the seat of the disease. Sympathetic nerve interruption is so far away from the true cause of Raynaud's disease that probably the operation eventually will be given up altogether for that condition when the etiologic factor is discovered.

NOTES ON THROMBO-ANGIITIS OBLITERANS

Buerger's disease is one of the most terrible afflictions which may beset a young man. It disables by pain, a fact which is too infrequently appreciated. A patient with Buerger's disease may be able to walk six blocks and then have to stop because of the pain (intermittent claudication). Even with this much vascular reserve, the dorsalis pedis and posterior tibial arteries are pulseless. The claudication, however, is not what impresses the patient so much. It is the rest pain, the boring, aching, intense pain in the ball of the foot, in a toe or in the instep which annoys him and keeps him awake at night and which disables so much. He could work at many jobs with a claudication which stops him only after three or six blocks, but he can work at none with the rest pain which nightly keeps him awake.

Gangrene or impending gangrene is a late stage of Buerger's disease. Usually the patient has had the disease a year or two before this complication supervenes. In the earlier stages it is easier to accomplish much more for the patient. For example in three cases these were the results:

A man, age 50, could slowly walk three blocks without claudication. Ganglionectomy not only abolished his rest pain, but increased his vascular reserve so that he can walk ten blocks.

A man, age 37, could slowly walk six blocks without claudication. Pain at rest was considerable and he had a small gangrenous spot on the plantar surface of his foot. Bilateral lumbar sympathectomy increased his vascular reserve so that he could walk sixteen blocks. The rest pain disappeared and the gangrenous spot healed.

A man, age 52, who had lost his left leg from gangrene two years previously, could walk, using his artificial leg, one-half block before claudication in the remaining leg stopped him. He had severe rest pain which made him accept sympathectomy. The operation relieved his rest pain, but increased his vascular reserve only so much that he could walk one block.

In each instance the foot pulses were absent, color changes, and other objective evidence for Buerger's disease were present. The first two were early, the last was a late stage of Buerger's disease. All three patients returned to work. If the vascular reserve was roughly measured in the distance walked without claudication, it was increased two and one-half times in the patient with early thrombo-angiitis obliterans, but less than twice in the one with late Buerger's disease. In the early cases, however, the increase was appreciable, seven blocks and ten blocks, whereas in the late case the improvement afforded a very short increase in distance. Such good results in my experience persist, if the patient's cooperation in not smoking is obtained.

It is my impression that Buerger's disease runs a self limited course. The organic changes seem to progress for two or three years then the disease stops and does not progress or proceeds at a much diminished rate. This is encouraging because if the patient can be tided over this phase with minimum organic changes in his vessels, he may then continue to lead a comfortable, useful, though a restricted, life.

Numerous drugs have been tried in this condition, few have given any definitely convincing evidence of their therapeutic value. Working on the theory that the disease so rare in women, might be favorably influenced by estrogenic hormone, I have used stilbestrol administered orally in daily one milligram doses in six cases. My impression is that it helps in combating the progress of the disease. It seems to retard the disease. None of these

FIG. 2.—Fibers of the internal oblique muscles are partially severed and partially separated. Those of the transversalis muscle are spread.

patients have developed gangrene and all are returned to a useful working life. All had stopped because they could not work. In each instance, however, the treatment included other measures and in each patient sympathetic nerve surgery.

The indications for operation on the sympathetic nerve system in thromboangiitis have never been standardized. Brown and Adson, in 1926, attempted to show a workable standard equation by comparing the rise in the skin temperature of the extremity with rise in body temperature induced by

intravenous injection of typhoid bacilli. I have measured the temperature of the skin at various places on the leg and foot and of each of the toes daily following ganglionectomy. Toes which were colder before operation became 4° C. warmer on the side operated upon. When the patient developed fever to 102° F., the toes on the side operated upon remain 4° C. warmer than those of the opposite foot, though the temperatures of each side had increased. This showed that even in fever the vasoconstrictor factor was present on the unoperated side and aside from the fact that the vasomotor index is no longer determined because of the disadvantages of the febrile reaction it did not show a true release of vasoconstriction as was formerly thought.

Various measures have been advocated for testing the vasomotor release expected from ganglionectomy usually by skin temperature studies and oscillometer studies after injecting peripheral nerves and infiltrating sympathetic ganglia as advocated by White. Though other ways such as warming the body, spinal anesthesia and novocain block of mixed nerves have been used to cause vasodilatation the most accurate test seems to be paravertebral sympathetic block and measurement of the skin temperatures by a thermocouple. My experience is that the oscillometer is of no value and that far from being an instrument of precision, it is never able to measure the minute increments of variation found after sympathetic nerve blocks in Buerger's disease. On the other hand, thermocouple readings before and after sympathetic nerve blocks may predict with fair accuracy this change to be expected after ganglionectomy. If a rise of 4° C. is obtained after sympathectomy comparing this figure with the preoperative rise after sympathetic blocks will usually show that the blocks predicted the postoperative temperature within from 0.5° to 2° C.

In measuring comparative temperatures of the skin by thermocouple, it is essential to employ definite areas on the thigh, leg, ankle, foot and on each toe. The measurements should be bilateral. The toes will show the greatest increment of temperature change. Their temperature may rise 3° C. while the leg rises 1° C. The greatest increase after nerve blocks will occur in from 20 to 30 minutes. Increases of 5° C. may be seen, but rises of even less than 0.5° C. do not mean than ganglionectomy will not be of value. A patient who had severe thrombo-angiitis obliterans, had a rise of only 0.25° C. in the toes following two sympathetic nerve blocks. Measurements made on the eighth postoperative day showed the toes to be 2° C. warmer than they were at the same room temperature prior to the operation. Even though very little increase in actual temperature of the extremity is obtained by operation —which would indicate a disappointing increase in the blood supply to the extremity—much is gained in pain relief for here again the surprising benefit of pain mitigated or abolished by sympathetic nerve section has no experimental proof but clinically it works.

Upon what then may the indications for operation (sympathectomy) in Buerger's disease depend? Upon the age and the condition of the patient. Sympathectomy offers so much to the early case that it seems always justified when the diagnosis is made in young people. The pulses in the foot however,

must be absent to justify a certain diagnosis. All patients under 50 years of age deserve operation unless gangrene makes the leg untenable and all patients between 50 and 60 deserve careful clinical consideration and an attempted evaluation of the expected improvement by sympathetic nerve blocks. Even patients aged 60 years, and over, have surprising vascular resiliency sometimes showing at age 65—2° C. rise in the temperature of the toes after sympathetic nerve block. Even though it is not justifiable to omit reasonable

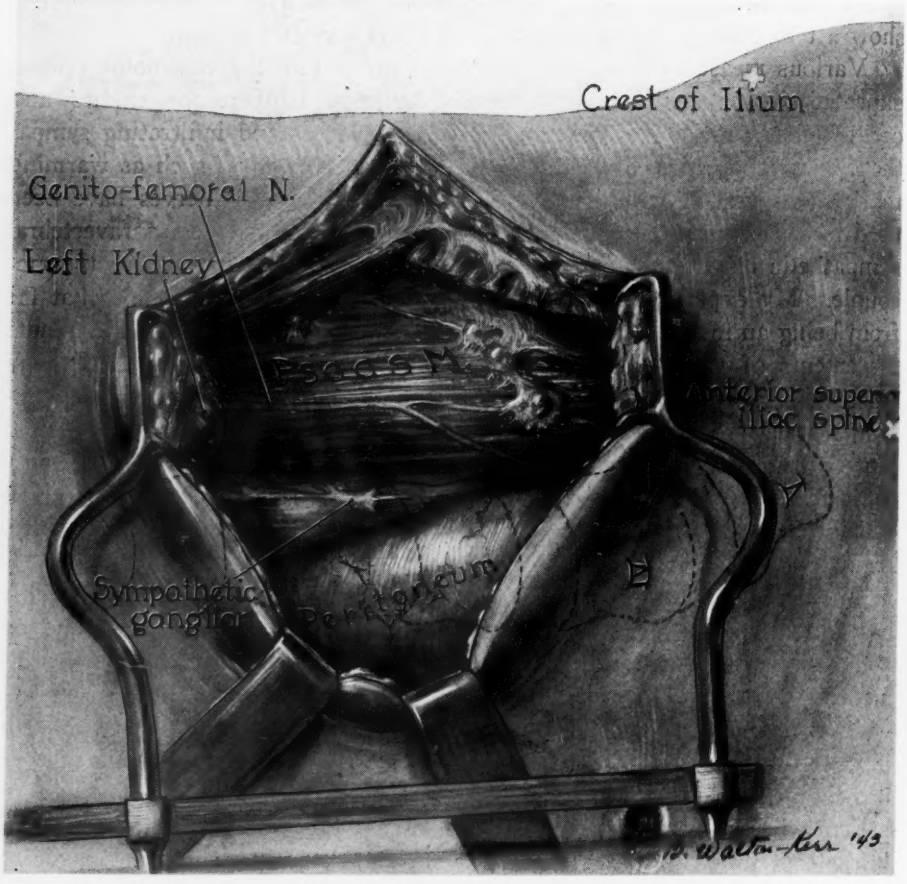


FIG. 3.—After pushing the peritoneum downward and forward, the ganglionated trunk is located in the valley between the vertebral body and the source muscle. Needles placed as for lumbar sympathetic block prior to the operation served as landmarks of the first interspace and fourth interspace. The lumbar chain is removed from the second through the fourth lumbar segments.

preliminary tests, the real bases of judgment for operation will be the age of the patient and the clinical appearance. It will almost invariably turn out that patients under age 50 deserve operation clinically; not just because the tests show favorable responses. The tests at this age show favorable responses because conditions are such the patient deserves operation. Those patients aged between 50 and 60 years frequently deserve operation and those over 60 seldom deserve major surgery for this condition.

Another reason why this is a safe corollary is the variation in the severity of the disease. In very young men the disease runs a violently progressive course. The intensity of its progress abates as the patient becomes older and should the disease make its first appearance in the sixth decade it is apt to be very mild and soon stops after a short active course.

All of this series of nine cases of Buerger's disease operated upon returned to work. None lost an extremity after sympathectomy. All were considerably disabled before operation.

SUMMARY

Sixty patients with conditions requiring temporary or permanent interruption of the sympathetic nerves, have been admitted to my private services at Touro Infirmary and the Baptist Hospital. Twenty-two operations for permanent section of the sympathetic nerves have been done in this group.

Pains influenced by interruption of sympathetic nerve function are varied. They occur in vascular disease with associated vasospasm and may be attributable not only to ischemia, but also directly to the vascular spasm. Vascular changes occur and persist after trauma; and pain associated with these may prolong disability unduly. These pains may be surprisingly relieved by paravertebral sympathetic nerve blocks permitting more active physiotherapy for mobilization of joints. Such repeated nerve blocks may often shorten a convalescence dramatically. Cases are presented which illustrate this.

Sympathectomy is indicated in all patients with thrombo-angiitis obliterans under age 50. The operation gives many times more increase in vascular reserve in patients with early Buerger's disease than in those affected with a late stage, for which it is too frequently reserved.

Raynaud's disease is not a fault of the sympathetic nervous system. Sympathectomy for it is frequently disappointing and eventually may be discarded entirely. These and other phases of the rôle of the sympathetic nervous system in vascular diseases were discussed.

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DISCUSSION.—DR. MARGARET STANLEY-BROWN, New York City: I would like to discuss Doctor Mahorner's paper. At the Goldwater Memorial Hospital in New York we see a large group of peripheral vascular cases. We have done high saphenous vein ligations on six cases of Buerger's disease, with complete relief of pain and increased exercise tolerance. The first case was done more than a year ago. We ran a parallel group of arteriosclerotics with no relief of pain.

DR. JOSEPH E. J. KING, New York City: All of us who were fortunate enough to have had charge of a Neurosurgical Service in the First World War had about the same experience; I cannot go into the details.

One of our most serious complications was causalgia. In some instances causalgia could be relieved by neurolysis, but in others neurolysis did not relieve the condition. Our best results in nerve suture were obtained after suture of the two main branches of the ulnar nerve in the region of the hypothenar eminence, and our worst results were in cases in which both the median and ulnar nerves were sutured, and in which there was gross damage of the major vessels.

An average of 65 per cent in return of function would be considered good. In some patients we had good results and in others the results were unsatisfactory. We found it was best to suture a nerve directly end-to-end without tension, with proper approximation, and with the site of the suture in good tissue. In some cases transposition of the nerve was useful. We have never seen good results following the long trellis transplant of nerves, probably due to the fact that in the patients in which these transplants were employed, a considerable portion of other soft parts was likewise destroyed.

DR. JAMES M. MASON, Birmingham, Ala.: Just a word in regard to causalgia. I recently saw a lady upon whom an intravenous injection had been attempted, but some of the solution had been placed outside the vessels, and a severe cellulitis had developed in the region of the bend of the elbow. Causalgia followed and periarterial sympathectomy had been done without relief. The painful symptoms were referred to the distribution of the median nerve. With novocain sympathetic block, the pain in the hand was relieved, and alcohol injection was immediately carried out. She was entirely free from pain when I had the opportunity of examining her four months later.

If I did not misunderstand Doctor Elkin, he does not approve of transvenous arterial suture in dealing with arteriovenous aneurysms, and considers that quadruple ligation and excision should always be carried out. In certain instances, transvenous arteriorrhaphy can be employed with advantage. It has a distinct place in dealing with arteriovenous aneurysms in the cervical region, where quadruple ligation and excision might be very dangerous.

DR. HOWARD MAHORNER, New Orleans, La. (closing): I was glad to hear Doctor Mason relate his experience with that particular patient, because it is the surprise that, at first, frequently comes with good results from sympathetic nerve block. In such cases there is a constant vasospasm and decrease in the temperature of the skin of that area. Possibly the spasm of the vessels has to do with pain. Leriche was the man who started such therapy in the last war, attempting to relieve these spasms by periarterial sympathectomy. There is no experimental proof from the lower animals for the relief of pain by this method. It must be done on human beings, and when it is done a few times with the result Doctor Mason described, one is convinced that it is the procedure of choice for such problems.

LT. COL. D. C. ELKIN, Atlanta, Ga. (closing): I am not a neurosurgeon. The neurosurgical part of the paper was written by Doctor Woodhall. Causalgia is best treated by neurolysis, sympathectomy, or both. Quadruple ligation is preferable to other methods, but I agree that, particularly in the neck, it may be dangerous, and transverse closure of the fistula may be the operation of choice.

THE SURGICAL TREATMENT OF EXPERIMENTAL COARCTATION (ATRESIA) OF THE AORTA*

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ACCORDING to Bramwell and King,[1] a pathologic narrowing of the aorta in the region of the isthmus, just beyond the origin of the subclavian artery, and varying from a slight degree to complete occlusion, is found in approximately one of every 1,500 autopsies. The mouth of the subclavian artery itself is affected in a small percentage of cases. Involvement of the isthmus itself between the left subclavian artery and the ductus arteriosus is known as the infantile type, while stenosis at or just below the ductus is designated the adult type.

No attempt will be made to review the extensive clinical literature on this subject. The large number of case reports in recent years indicates that the condition is not extremely rare. The chief physical signs consist of evidences of an increase in collateral arterial circulation and an increase in arterial blood pressure in the arms and a decrease in the legs. That coarctation of the aorta is not incompatible with prolonged survival is shown by the observation of Abbott[2] that the average age of patients at death is 36 years.

Recent experimental interest in coarctation of the aorta has followed the work of Goldblatt, and his associates,[3] on the production of hypertension by constricting the renal arteries. The effects of constriction of the aorta have been studied by a number of observers.

Halsted[4] unsuccessfully attempted complete occlusion by metal bands of the thoracic aorta of dogs. Matas and Allen[5] reduced greatly the caliber of the thoracic aorta by a method of plication or infolding of its walls. Total occlusion did not result. Reid,[6] and Pearse[7] blocked the thoracic aorta by the insertion of foreign bodies into its lumen. Pearse[8] subsequently reported successful occlusion by the external application of cellophane around the wall of the aorta. By the employment of rubber bands in multiple-stage operations, J. C. Owings[9] was successful in completely occluding and subsequently dividing the aorta in the thorax. His work emphasized the necessity for bringing about the occlusion gradually. In our preliminary studies we attempted to cause atresia of the aorta by a method similar to that used by Owings, but a good many animals were lost because of hemorrhage. In most instances this occurred at the time that an attempt was being made to tighten the band.

*Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

The problem considered here may be stated as follows: Since coarctation of the aorta may result in untoward incidents, such as cerebral accidents, it is desirable to find a method to circumvent the stenosis or atresia. It seemed possible that this might be done by performing an anastomosis between one of the large arteries which come off the aorta proximal to the constriction and the side of the aorta distal to the point of stenosis.

METHODS AND RESULTS

A total of 43 experiments was performed on dogs. This number does not include 23 earlier experiments in which an attempt was made to cause atresia of the aorta by the use of rubber bands. Thirty-three of the 43 animals were males. The weights of the dogs ranged from 9.6 to 18 kilograms.

The operative procedures were performed in the following manner: The animals were anesthetized by the intravenous administration of nembutal, 25 mg. per Kg. of body weight. An intratracheal tube was introduced through the mouth in order that air under positive pressure could be used. With aseptic technic an intercostal incision was made in the fourth interspace on the left. The pleural cavity was entered and a good exposure of the upper part of the thoracic aorta and the left subclavian artery was obtained. The left subclavian artery of the dog arises from the aorta as does that of man. Both carotid arteries and the right subclavian of the dog arise from the innominate artery. The mediastinal pleura overlying the left subclavian artery was incised and the thoracic part of the subclavian was freed from adjacent structures. A bulldog clamp was placed on the subclavian artery at its most proximal part. The artery was then ligated about 5 cm. distal to this point and was divided just proximal to the ligature. The aorta immediately proximal and distal to the origin of the left subclavian artery was mobilized sufficiently to allow two rubber-shod clamps to be placed upon it. These clamps were placed about 6 cm. apart and the aorta was completely occluded by each of them. The aorta was then divided midway between the two rubber-shod clamps and each end was closed by two rows of sutures of very fine silk. The first row consisted of mattress sutures which were placed about 5 mm. from the ends of the divided vessel. The second row was a continuous one which was placed at the points of division of the vessel. The ends of the vessel were anchored together in order that further separation might not occur. The importance of this point has been emphasized by Owings.[9]

A short transverse incision was made into the side of the distal aorta slightly beyond the point at which its end had been closed. The length of this incision was slightly greater than that of the diameter of the subclavian artery. It is important that this incision should be in a transverse direction because the wall of the aorta tears rather easily if the incision is in a longitudinal direction. Furthermore, it is important not to try to clean the aorta of adventitia as is usually done in arterial anastomoses. And end-to-side anastomosis between the proximal end of the subclavian artery and the side

of the aorta was then performed. As in all blood vessel anastomoses, the important point is that intima should be approximated to intima by eversion. A guide-suture was placed at the posteromedial angle. A continuous row of china beaded silk sutures was used for the posterior row. This was tied to a guide-suture which was placed at the posterolateral angle. The anastomosis was completed by an anterior row of continuous sutures of silk. The rubber-shod clamps were removed from the subclavian artery and the aorta. Slight bleeding at the site of the anastomosis could usually be controlled by light pressure for several minutes. In a few instances it was necessary to place an additional suture. A thrill was felt in the aorta distal to the anastomosis following the removal of the clamps. The incision in the chest wall was closed in layers with interrupted sutures. The steps of the operation are shown in Figures 1 and 2.

As stated previously, 43 such operations were performed. None of the animals died during the operation. The time period that the aorta was occluded during the performance of the anastomoses varied from 40 to 65 minutes. The mean arterial pressure (approximate) as measured in the femoral artery by the needle puncture method immediately after completion of the operation ranged from 40 to 130 Mm.Hg., the average reading being 85 Mm.Hg. The knee jerks were present and usually hyperactive when tested at the completion of the operation.

Only ten of the 43 animals survived the operation for several months or longer. Of the remaining 33 animals, the average length of life was six days. There were six deaths in the first 24 hours. Fifteen of the 33 animals lived for periods ranging from five to 25 days. Included among the causes of death were empyema, distemper, pneumonia, hemorrhage from the site of the anastomosis or the end of the aorta, thrombosis of the anastomotic site, pleural effusion, and paralysis of the posterior part of the body. In eight instances the cause of death was not discovered.

The predominating postoperative complication was paralysis of the posterior part of the body. Paralysis of varying degree occurred in almost half of the cases. In the majority of instances it was complete and was spastic in type. In a few instances there was simply weakness of the posterior extremities. The paralysis was usually noted during the first 24 hours after operation but in several instances it was delayed in onset.

Further description will be limited to the ten cases in which the dogs lived for several months or longer after operation. Nine of these ten animals were males, whereas 33 of the total of 43 were males. The mean femoral arterial pressure at the completion of the operations ranged from 65 to 130 Mm.Hg., the average being 89 Mm.Hg. The length of time that the circulation was interrupted during the operation varied from 40 to 50 minutes, the average being 44 minutes. These figures differ very little from those observed in the experiments in which survival did not occur. Six of the ten animals showed no paralysis at any time. Two of the dogs had a slight weakness of the posterior extremities which disappeared in a few days. There was a partial per-

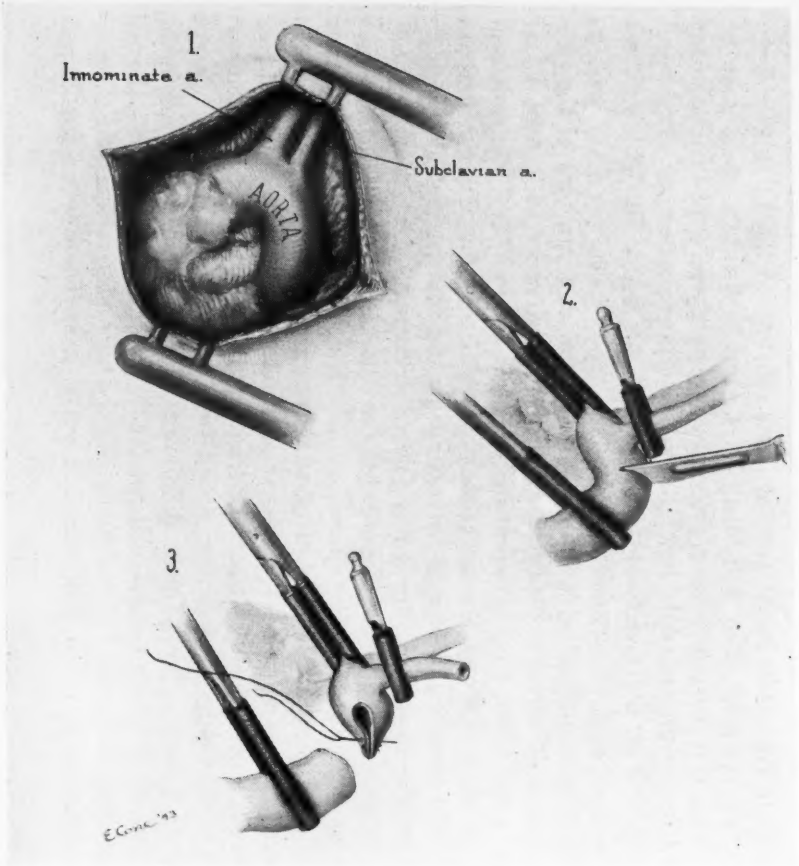

FIG. 1.—Showing the first three steps of the operative procedure. (1) Exposure of the aorta and left subclavian artery through an incision in the fourth left interspace; (2) occlusion of the aorta and left subclavian with rubber-shod clamps. Division of subclavian artery and aorta; (3) closure of divided ends of aorta.

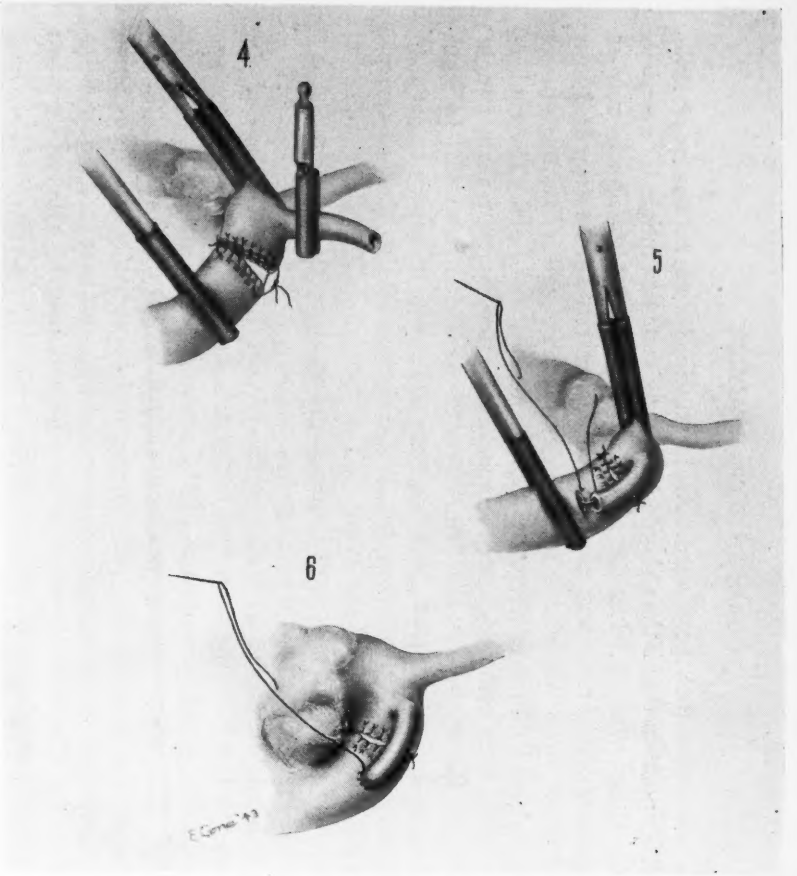

FIG. 2.—Showing completion of operation. (4) Closure of ends of divided aorta with two rows of sutures. Ends of vessel anchored in order that separation will not occur; (5) opening made in side of distal end of aorta. Beginning of anastomosis between end of subclavian artery and side of aorta; (6) anastomosis completed.

sistent paralysis of the posterior extremities in one animal and a delayed complete paralysis in the remaining one.

The left carotid artery of the animals that survived the operation for an extended period was exposed and was explanted beneath the skin of the neck in order that it might be punctured with a needle that was used for blood pressure determinations. The carotid and femoral arterial pressures were determined from time to time. In each instance the carotid arterial pressure was higher than the femoral but in no case was this difference very great. The figures are given in Table I. The flow through the subclavian artery and through collaterals was sufficient to prevent hypotension in the posterior part of the body.

TABLE I

THE ARTERIAL BLOOD PRESSURE OF ANIMALS WHICH SURVIVED FOR EXTENDED PERIODS THE OPERATIVE PROCEDURES

Exp. No.	Femoral B.P.* Immediately Postoperatively Mm. Hg.	Time Since Operation	Carotid B.P. Mm. Hg. Needle Puncture	Carotid B.P. Mm. Hg. Cannula†	Femoral B.P. Mm. Hg. Needle Puncture	Femoral B.P. Mm. Hg. Cannula†	Remarks
31	90	10 months	173		168		Spastic paralysis hind legs. Sacrificed.
36	80	10 months	175	174/146	145	146/128	Excellent condition. Sacrificed.
41	65	8 months	155	177/165	120	160/150	Excellent condition. Sacrificed.
44	130	10 months	150	190/168	135	186/156	Excellent condition. Sacrificed.
48	90	9 months	160		150		Excellent condition. Left subclavian artery ligated and divided. Died 24 hours later.
54	95	4 months	160		138		Died 128 days following operation. Cause undetermined. Probably pneumonia.
56	70	6.5 months	185	154/140	165	134/120	Hind legs semispastic. Sacrificed.
59	85	8 months	165		150		Alive and well.
65	90	6 months	135		130		Alive and well.
66	90	6 months	150		140		Excellent condition. Sacrificed.

*Needle puncture.

†Mercury manometer, no maximum-minimum valves. Determined under nembutal anesthesia shortly before animals were killed.

One of the ten animals died 128 days after the operation. This animal had appeared to be in good condition, and the cause of death was not determined with certainty but was believed to be due to pneumonia. The subclavian-aortic anastomosis was patent. The left chest of one animal was opened nine months after the original operation and the left subclavian artery was doubly ligated and divided. Paralysis of the posterior extremities developed and the animal died 24 hours following operation. This experience suggests that the transplanted subclavian artery had been effective in conveying blood to the posterior part of the body. Six of the remaining eight animals were sacrificed at intervals ranging from six to ten months following the performance of the subclavian-aortic anastomosis. Hill's radiopaque mass was injected into the arterial system and roentgenograms were made immediately after death. Typical roentgenograms are shown in Figures 3 and 4. The anastomosis was patent in all instances. The subclavian artery appeared

to be somewhat dilated. There was some increase in the size of collateral pathways but this was not pronounced. The roentgenograms shown in Figures 3 and 4 may be contrasted with that in Figure 5, which is reproduced from the paper by Doctor Owings. It will be recalled that he produced gradual occlusion of the aorta, and the tremendous increase in the collateral arterial

FIG. 3.—Reproduction of roentgenogram which was made following the injection of Hill's mass. The arrow points to the left subclavian artery, which was used for anastomosis to the side of the aorta distal to the point of its division and closure. The collateral arterial pathways are only moderately enlarged.

pathways is shown in Figure 5. Photographs of two of our gross specimens are shown in Figures 6 and 7. Microscopic sections of liver and kidney did not reveal abnormalities. Photomicrographs of the vessels at the site of the anastomosis are shown in Figures 8 and 9.

Two of the animals are alive and appear to be entirely normal. They will be observed for an extended period.

DISCUSSION

It had been demonstrated previously that dogs will not survive an acute one-stage occlusion of the thoracic aorta. It was found[10] that the survival period following one-stage complete occlusion of the thoracic aorta is usually only about three hours. Erlanger and Gasser[11] conclude that the fall of blood

FIG. 4.—Roentgenogram following the injection of Hill's mass. Arrow points to left subclavian artery.

pressure and early death after occlusion of the aorta are due to a reduction in the effective volume of blood. The fact that some of the animals in the present study survived complete division and closure of the aorta shows that the subclavian artery was effective in conducting blood to the posterior part

of the body. The fact that there was not hypotension in the posterior part of the body and hypertension elsewhere also suggests that the blood flow was adequate. The absence of a decided increase in the size of collateral pathways (as demonstrated by Owings in simple progressive occlusion) indicates that

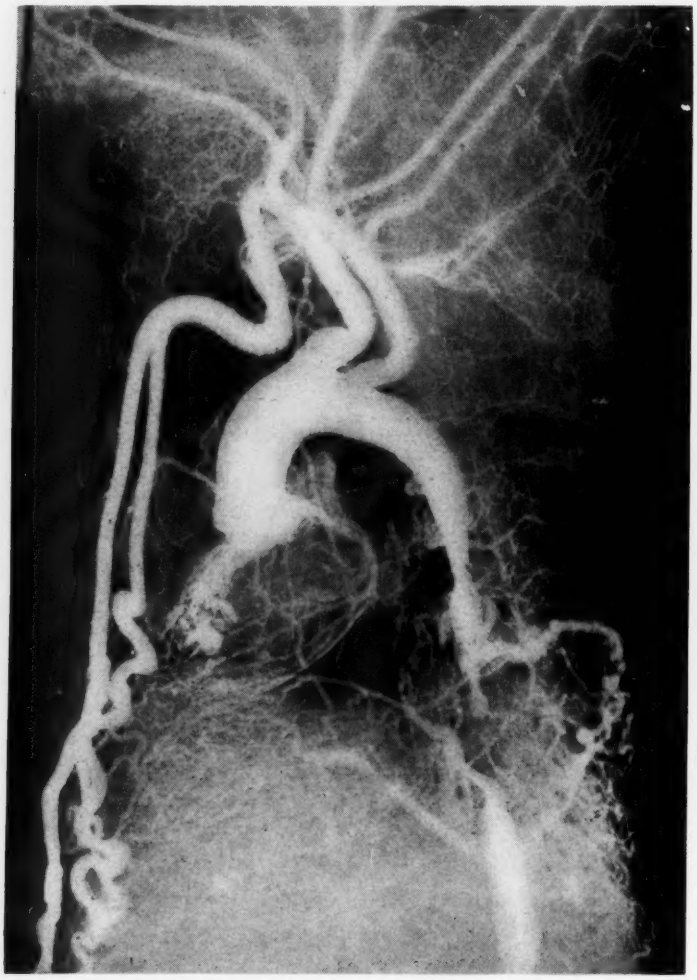

FIG. 5.—Roentgenogram reproduced by courtesy of Dr. J. C. Owings (Annals of Surgery, 115, 596, 1942). The extensive collateral circulation which develops as a result of multiple stage occlusion and division of the thoracic aorta is demonstrated. The collateral circulation is much less extensive in our studies (Figures 3 and 4) in which a subclavian-aortic anastomosis was performed.

in our studies a large part of the blood supply to the posterior part of the body must have passed through the subclavian artery. The death of one animal following subsequent operative occlusion of the subclavian-aortic anastomosis is also evidence in favor of this viewpoint. Our studies show that the femoral arterial pressure at the conclusion of the operation does not allow one to predict with any degree of certainty the chances of survival of

the animal. Some of the animals with the highest femoral arterial pressures died within a relatively short time after operation.

In only five of 43 experiments was the subclavian artery or the anastomosis blocked by a thrombus. In two of these there was a clot in the proximal end of the divided aorta which extended beyond and blocked the mouth of the

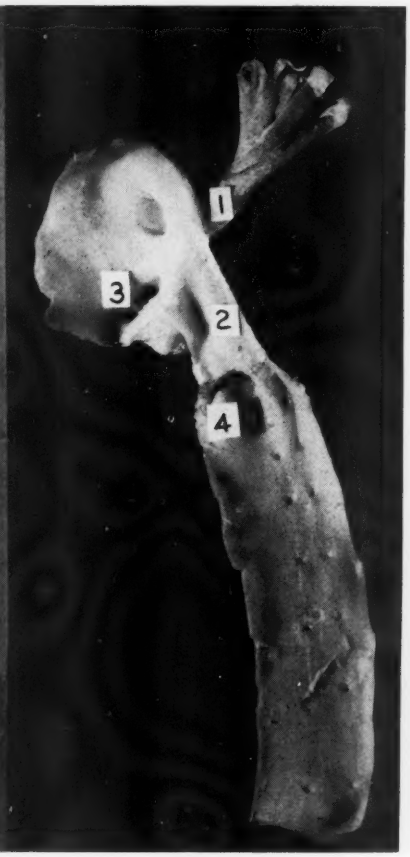

FIG. 6 FIG. 7

FIG. 6.—Showing a photograph of the thoracic aorta which was removed 10 months after we had divided the aorta and had performed an anastomosis between the subclavian artery and the side of the distal end of aorta. (1) Innominate artery arising from the arch; (2) left subclavian artery; (3) proximal end of divided aorta; and (4) distal end of divided aorta.

FIG. 7.—Showing a specimen of the aorta which was opened in a longitudinal direction. (1) Innominate artery; (2) left subclavian artery which is considerably larger than at the time that the anastomosis was made; (3) proximal end of divided aorta; and (4) distal end of divided aorta.

subclavian artery. Thus, the anastomosis appears to be feasible from a technical standpoint.

The most disturbing factor was the paralysis which resulted in many instances. The cause for this has not been determined and the subject merits further study. It seems most likely that the paralysis results from an interference with the blood supply to the spinal cord. In a description of the results of complete and incomplete occlusion of the abdominal and thoracic

aorta by metal bands, Doctor Halsted[4] states: "The study of the spinal cord was entrusted to Mr. P. K. Gilman (now Doctor Gilman, and a member of

FIG. 8

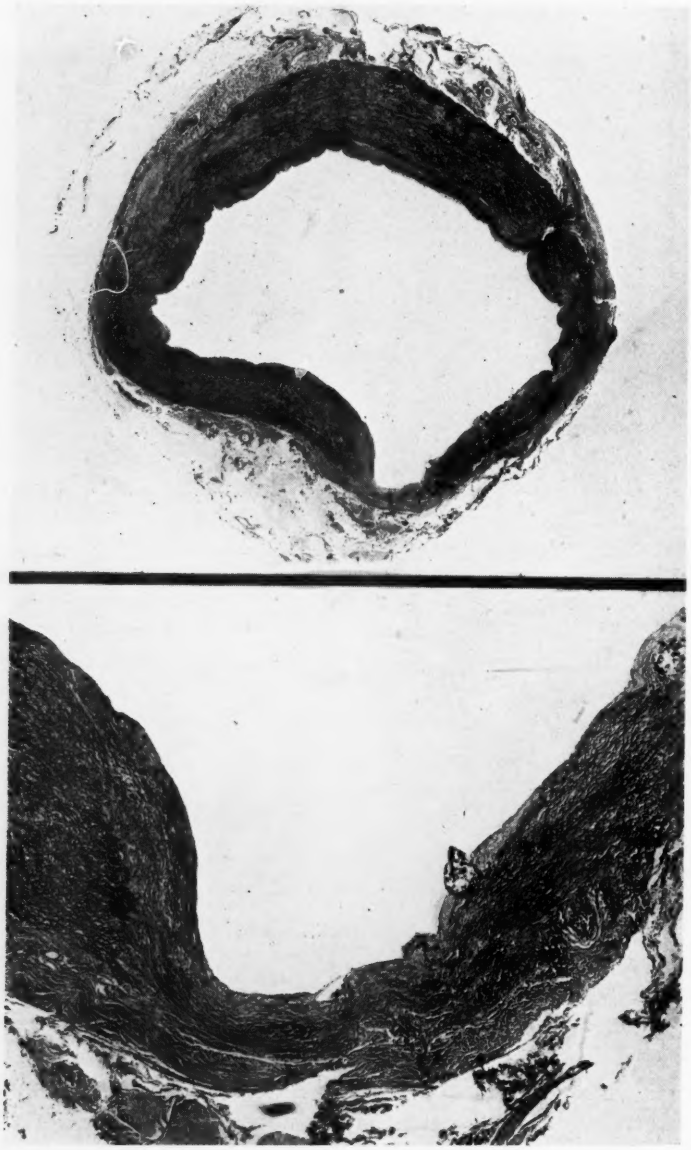

FIG. 9

FIG. 8.—Photomicrograph of site of anastomosis of end of subclavian artery to side of aorta. (×8)

FIG. 9.—Photomicrograph of site of anastomosis. (×35) Several silk sutures are visible.

my staff), whose trained eye discovered in a number of cases, about three months after operation, a deposit of extradural fat about the cord below the

site of the aortic band. In three cases the production of fat was so great that it filled, seemingly under considerable tension, the vertebral canal." This finding was not encountered in our studies. Furthermore, the onset of paralysis was so rapid in some of our animals that it could not be explained on this basis. In two control experiments the thoracic aorta and the left subclavian artery were occluded for 50 minutes and then released, without division and anastomosis, and paralysis did not occur.

There would be some obvious objections to the use of the subclavian artery in man if the operative procedure described here, or a modification of it, was to be used. In the first place, the subclavian artery is the most important vessel in supplying blood to collateral pathways around the point of stenosis or atresia of the aorta. Division of this vessel might interfere considerably with this blood supply. In the second place, division and rotation of the subclavian artery might interfere too greatly with the circulation of the arm. It would seem that the left common carotid artery is a better vessel to use. It was not used extensively in the present experiments because it arises in the dog from the innominate artery rather than from the aorta and because its lumen is smaller than that of the subclavian. The left common carotid artery was anastomosed to the aorta in several dogs, but the femoral pressure immediately afterward was found to be less than 50 Mm.Hg. and death occurred. The common carotid artery presents the advantage over the subclavian of having few branches. Since many collateral pathways are already existent in the patient with coarctation, it is our impression that the additional quantity of blood supplied by the carotid would be sufficient to improve considerably the circulation of the lower part of the body.

Although this operation has not been attempted in man, it would appear that the enlarged collateral vessels which enter the aorta below the point of the stenosis in the patient with coarctation would make it difficult to obtain a dry field for the performance of the anastomosis. On the other hand, it would appear that the patient with a chronic partial occlusion of the aorta would tolerate a temporary complete occlusion better than the normal dog in which there has been no stimulus to the formation of collateral arterial pathways. In any case, this procedure, or a modification of it, should be considered only in those cases of coarctation in which the outlook is very grave since many patients with coarctation of the aorta have a fairly long life expectancy.

SUMMARY

These experiments were undertaken with the idea of devising a means for the surgical treatment of coarctation of the aorta. At a one-stage operation the thoracic aorta was divided, the two ends were closed, and the proximal end of the divided left subclavian artery was anastomosed to the side of the aorta beyond the point of its division. Some of the animals survived this procedure, and there was evidence of adequate blood flow to the posterior part of the body. The possible clinical application of these studies is discussed.

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LIGATION OF THE ABDOMINAL AORTA FOR ANEURYSM*

COMPLICATED BY RUPTURE INTO THE RETROPERITONEAL SPACE

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THE TREATMENT OF ANEURYSM of the abdominal aorta by ligation was thoroughly reviewed in 1940 by Bigger, Elkin, and Matas. There have been no reports of this procedure subsequent to that date. These authors found that since the original report of Cooper, in 1817, to their own, in 1940, there had been only 30 reports of ligature of the abdominal aorta for aneurysm of the aorta or of the common iliac at its origin. Only seven of these operations could be regarded as in any degree successful. Elkin included four instances in which the aorta had been ligated for trauma or tumor and only one of these patients lived for any length of time afterwards.

Bigger claimed that many of the problems connected with aortic aneurysm could be solved only by observation on patients thus afflicted. Consequently, it seemed important to record the results of ligation of the abdominal aorta in a patient with aortic aneurysm and rupture into the left retroperitoneal lumbar space.

Case Report.—Hosp. No. 581: J. G., male, age 52, was admitted to the Strong Memorial Hospital January 10, 1942. He was feeling about as usual until the previous day. During the morning he began to have dull pain in the left flank. He ate lunch downtown and the pain became severe enough to make him go home to lie down. At 2:30 P.M., he had a sudden agonizing pain in the left lower quadrant. When his physician saw him ten minutes later, he was in obvious shock, with a greenish pallor, cold sweating, and a small thready pulse. He was groaning with pain and afraid to move lest his pain become worse. The pain did not radiate. The whole left side of his abdomen was tender. There was slight tenderness over the left kidney region. The muscles just above the pelvic brim on the left were held slightly tensed but the right side of the abdomen had neither muscle spasm nor tenderness. It required two ½ grain doses of morphine to get relief during the next 30 minutes. In 1½ hours, his shock was under control, his pulse was much stronger and he could be moved without much pain. He remained free of pain till about 12 hours later. Then (1:30 A.M.) the pain returned to the same region with lessened intensity. Morphine gr. ½ and atropine gr. 1/150 gave relief, so that he slept for 1½ hours. At 3:00 A.M. he had more pain, and more heavy drugging which gave him only about four hours relief. At 8:00 A.M. he was brought into the hospital by ambulance. He had not voided for 18 hours.

In the past, he had had severe episodes of migraine, due in part to food sensitivities. There were also infrequent asthmatic attacks, especially with respiratory

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infections. He had been away from work for about one year (till six months ago) because of marked depression. He had had his appendix removed 23 years previously. He had had a slow tremor of the hands for years.

Physical Examination.—The patient was pale. There was tenderness in the left lower quadrant and resistance to deep palpation. A definite, exquisitely tender mass could be felt in the left lower abdomen. Ballottement was not attempted because of the pain. There was only slight costovertebral tenderness. His temperature was subnormal, pulse 100, respirations normal. Blood pressure 120/70.

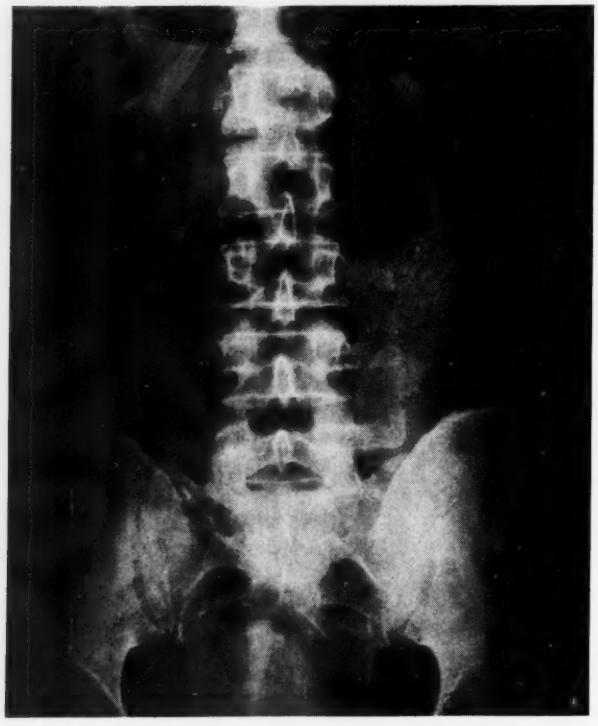

FIG. 1.—An intravenous pyelogram shows slight displacement of the left ureter and a normal kidney pelvis. The calcifications to the right of the fourth lumbar vertebra and in front of the sacrum were considered as possibly arteriosclerotic.

Laboratory Data: Red blood count 3,200,000, hemoglobin 8.5 Gm., white blood count 13,800. The urine showed no blood and a good function as judged by the phenolsulfonphthalein test. The stools were not remarkable. The nonprotein nitrogen was 61 mg. per cent, creatinine 2.1 mg. per cent, total proteins 5.7 per cent, phosphorus 8.1, and phosphatase 3.7 Bodansky units. The blood Wassermann was negative.

He was a diagnostic problem at this time. Ureteral stone was suggested but ruled out by the negative urinary examination and the negative intravenous pyelograms (Fig. 1). Other possibilities were retroperitoneal tumor and dissecting aneurysm. These were thought of because of some calcifications seen in all films both to the right of the spine at the level of the iliac crest; and across the sacrum from right to left, as well as a large soft-part shadow in the left lumbar region.

On bed rest he appeared to improve. During the first days in the hospital he had the sensation of something pressing in his abdomen. He showed some dullness over the lower half of the left chest with diminished breath sounds and diminished whispered voice. The blood pressure in both arms was 120/78. The tenderness gradually subsided

during the first week. It was possible to feel a smooth mass which extended toward the midline but had no definite edge. The left femoral artery seemed to have a smaller excursion than the right.

Blood pressure, right thigh 130/80, left 140/82 (not entirely satisfactory). By the end of the first week, there was an easily distinguishable pulsation over the left lower quadrant. This was synchronous with the aortic pulsation. It was not possible to make out expansile pulsation in the mass. On the twelfth day after admission there

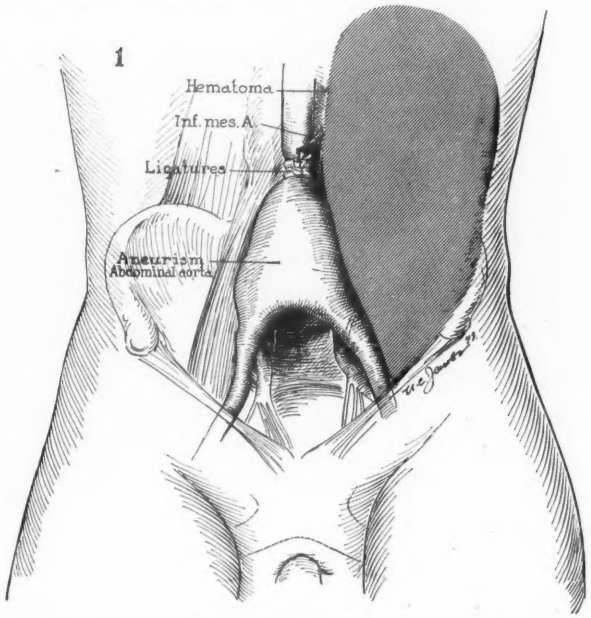

FIG. 2.—The condition following operation showing the blood extravasation into the left lumbar retroperitoneal space. The double ligation of the aorta with cotton tapes below the origin of the inferior mesenteric artery is shown.

was noted a definite swelling of the left thigh with slight pitting edema at the ankle. There were no signs of thrombosis, such as Homan's sign, tenderness on calf pressure or tenderness in the popliteal space. He began to sit up on the edge of the bed about this time. Things seemed to be progressing nicely. He was up in a chair for a short time on the fourteenth day after admission.

On the morning of the next day, he began to have aching in the left thigh, gradually extending to the leg as well. The entire limb was more swollen. There was no tenderness except in the deep femoral triangle. The peripheral arterial pulsations were easily made out.

A venogram was made because of a slowly rising pulse rate. It showed presumable thrombosis in the left femoral vein. Exploration of the abdomen was done in the hope that a diagnosis of the mass could be secured as well as ligation of the iliac vessels. There was a large mass with expansive pulsations in the left iliac fossa. The aorta above this contained several "egg-shell" plaques. There was a large area of blood clot outside the left iliac aneurysm extending as far upward retroperitoneally as the left kidney. Closure was made, as this problem required discussion with the patient and his family.

Lumbar sympathetic block was done four days later, without untoward effects. No definite improvement was noted.

On January 29, 1942, the abdominal wound was reopened and two tape ligatures, about one-half inch apart, were placed about the terminal aorta just below the inferior

mesenteric artery (Fig. 2). They were tied so that a faint pulsation could still be felt in the dilated abdominal aortic aneurysm. The expansile pulsation disappeared and the area became soft. The operation caused little disturbance in the patient's general condition. On recovery from the ether anesthesia, he had severe pain in his left leg. This was relieved by lumbar block in the second interspace. There was also abdominal and back pain, and later, pain in the left hip region. The next morning, the left upper leg became mottled purplish-red. Blisters appeared over the lower part of this limb. The right leg seemed normal but the left was quite swollen. Two days after operation,

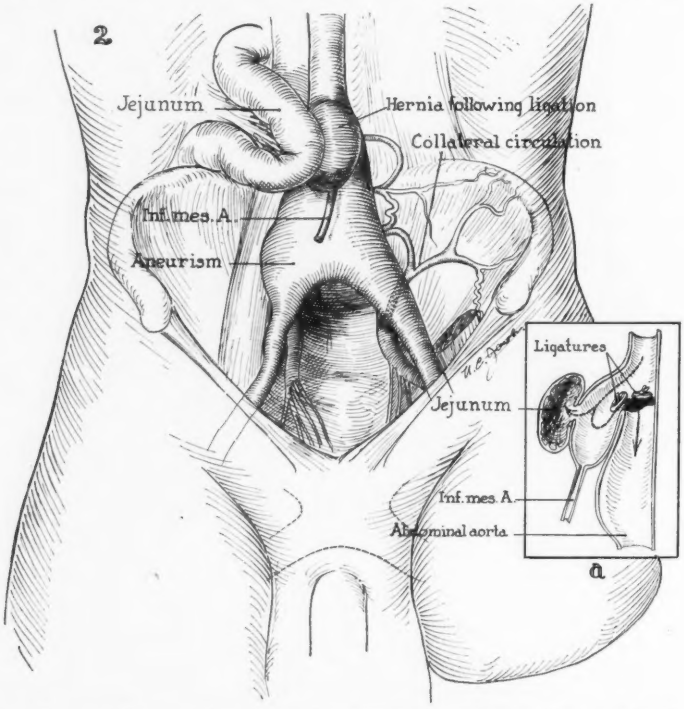

FIG. 3.—The condition found at postmortem examination. The collateral circulation was worked out by dissection and studies of the injected vessels in roentgenograms.

he moved his right leg and foot without trouble. There was a good capillary return on pressure over the skin. The circulation seemed fair but no pulsations could be recorded in the peripheral vessels. Sensation was not present. The toes of the left foot began to shrivel. On February 1, 1942, tingling and returning sensation was noted in the right leg. The left leg began to show a line of demarcation at the midthigh level. A good femoral pulse could be palpated on the right on February 4.

He began to have evidence of absorption from his gangrenous leg by February 8. A high thigh amputation was performed on February 9, under sodium pentothal anesthesia. There was very little bleeding. The main arteries and veins were completely occluded by clots. All muscular tissue was excised until free bleeding was obtained. Many of the muscles were necrotic all the way to their attachments at the pelvis. The wound was blown full of sulfadiazine and left open except for one suture in the center of the stump which brought the skin together there.

The stump healed slowly. The wound was sluggish and reactionless. It oozed a thin serous material and became covered with dirty sloughs. *B. subtilis* became established in it in spite of every precaution. Sulfadiazine was used at first, then azochloramide and normal saline. By the first of March, granulations were healthy appearing, the

stump was shrinking in size, and progressing favorably. He began to complain of a phantom limb. He noted pain in it "as if the heel were held in a vise." The pain started usually at 6:30 P.M. and lasted fully two hours. Another time when the phantom was especially prominent was just after he awakened. The limb seemed of normal length. The toes were not in an unusual position. The consciousness of the limb disappeared as he tended to move about. By March 11, the epithelium was growing across the stump end. It was healed except for the inner side. He was walking about his room on crutches. He felt no pain in the stump now but he was conscious of the phantom at times. Pain in this was now mostly confined to the foot though he had a

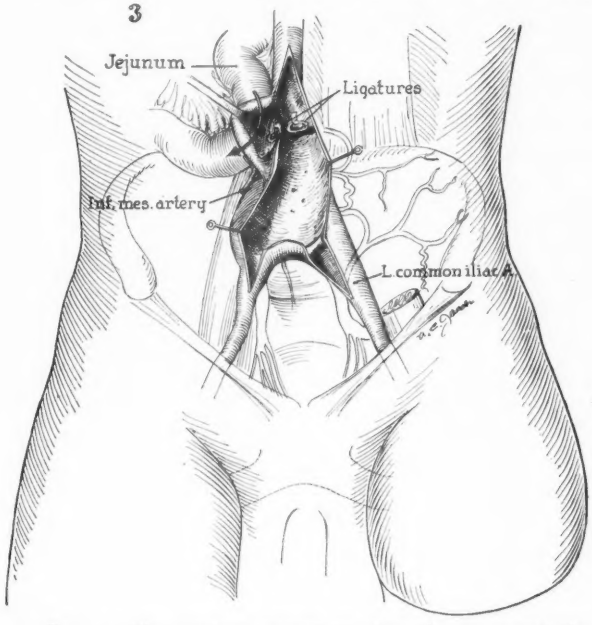

FIG. 4.—The specimen opened to show the tapes and the fistula to the jejunum from the newly formed aneurysm of the aorta.

sensation of constriction in the calf now and then. The foot seemed bent at an angle. On March 17, he had severe pain in the phantom limb. The pain shot through his foot "like a red hot iron from heel to toe." There was a sensation through the instep "as if it is being pushed together,—a poor description but it is hard to say exactly what it is like,—a squeezing sensation." There had been also a flickering sensation down the back of his thigh "sort of like a fork of lightning." He was conscious of no sensation between the stump and the foot. The limb had shortened to the level of the knee. The foot was pointed inward. The toes seemed to be bent upward. The recent bad weather caused accentuation of all these complaints. Codeine gave relief. A paravertebral block gave relief for about one-half hour. The stump slowly progressed toward healing. The phantom was shrinking as well so that by March 24 it was above the knee level. There were occasional stabs of severe pain which caused him to wince and even to cry in agony. These pains began to space out and were less intense during the last three days. On April 2, three small pinch grafts were taken from the skin of the lateral stump under local procaine anesthesia and applied to the remaining granulating surface. While the grafts were being taken, he claimed that the pain in the phantom foot was increased although he felt nothing at all locally. He was discharged on April 10, 1942, with the stump practically healed. He was getting about on crutches. The phantom limb still "gave him hell for one to two hours a day."

At home he gained in weight and strength. He was able "to walk a mile on crutches." His appetite and digestion were good. Stools were normal. The phantom leg still gave trouble. It had shrunk in size and occupied a position with the knee flexed and the foot opposed to the right thigh. There was continuous prickling in the foot and frequent attacks of "red hot" pain in the dorsum of the foot and in the skin. When he first went home he had two episodes of pain daily. One came in the late afternoon and the other in the evening. Gradually the afternoon attack came earlier

FIG. 5.—Injection of the artery with a radiopaque liquid latex showed an extension into the jejunum which was explained by later dissection.

and earlier in the day till it finally came on just after breakfast. Walking and exercise caused the morning attack to lessen but the evening episodes became worse. Deep pressure on the stump produced pain only in the stump. But light stroking of the skin in the posterior flap produced severe pain and burning referred to the toe area of the phantom foot. The sensitive areas were especially marked over the medial third of the posterior flap of the stump with a less sensitive area over the outer third. He got relief on "Alba D"—a morphine replacing substitute drug. He had returned to work, taking part in committee and departmental duties. He carried on his dictation and correspondence as he had previously.

He was again admitted to the hospital, and a spinal anesthesia was done. Anesthesia was obtained to level of D9. Motor function of the right lower leg was lost for ten

minutes. All painful sensations were absent in the left stump but he retained sensation in the phantom limb—though it was not a disagreeable type of sensation. Stroking of the trigger areas in the skin was not felt and no psychic disturbance was elicited. It was decided that section of the peripheral nerve trunks might help in reducing his pain. Accordingly, under avertin and nitrous oxide-oxygen anesthesia, the great and lesser sciatic nerve stumps were divided. The amputation stump had very little blood supply.

After he left the hospital, the phantom leg pain was reduced at least 90 per cent. Most of the time pain was entirely absent. The phantom itself continued to be present. It gradually became smaller, with the leg flexed on the thigh and the heel drawn up into the crotch. He had the sensation that he could straighten out the leg by great voluntary effort but it would snap back promptly into the flexed position. He was rarely able to forget its existence.

FIG. 6.—The circulation in the right thigh was in excellent condition.

He went into a depression fearing that he would never be able to undertake his previous occupation. When he did his scholastic work, he found it hard to concentrate. He made frequent errors in mathematics which distressed him as he had always been accurate. He began to take relatively large doses of bromochloral and phenobarbital to get relief from insomnia and worry. After July 1, he was found to have a bromide level of 210 mg. per cent in the blood. Bromochloral was stopped. Seconal and increased phenobarbital was substituted. The mental confusion cleared up and the blood bromide level dropped to 75 mg. per cent in a week. Large doses of sodium chloride had been used but in spite of this the bromide retention had gone on.

Early in July he had an attack of severe diffuse, lower abdominal pain. The pain did not localize. It was accompanied by sweating, pallor, a drop in blood pressure from 110 to 100, and a sensation of fright. Two doses of ¼ grain morphine gave relief. In 24 hours, he seemed normal again. Two similar attacks came at two-week intervals with rapid relief following morphine. Between attacks he walked about with crutches up to a mile a day. He drove his car to his office daily where he took care of his correspondence. One of the attacks came a couple of hours after he had climbed a flight of stairs.

His depression deepened. Reassurance was of value for only a day or two. He was convinced that he could never carry on his duties. On two occasions he stated that he would end his life if the means were at hand.

While taking a walk on August 14, he had a mild attack of abdominal pain. It persisted in varying intensities up to the time of his death. There was no let-up in it even under heavy sedation. He complained of a "peculiar sensation in the head." By August 16, the pain had become very severe, accompanied by pallor, sweating, and a rapid, weak, irregular pulse. He was quite weak and unable to eat solid food. On August 18 he passed by rectum a large amount of fluid containing both dark and bright red blood. During the day, he had two more similar movements. He was extremely pale and very weak by nighttime. His pulse increased to 130, his blood pressure dropped to 90/50. There were several attacks of hiccoughs lasting as long as one-half hour. He appeared to be on the point of death. But on August 19, he sat up on the side of

his bed and washed himself. He was quite confused mentally during the next day or two. On August 22, there were two more blood-containing stools. Hiccoughing continued, relieved somewhat by various sedatives. He lived on liquids mainly. He went on in this manner until 11:00 P.M. on August 28 when he quietly expired. This followed another painful episode associated with sweating, pallor and failing pressure. During all these attacks of abdominal pain, there was no spasm of the abdominal muscles. Even fairly deep pressure did not alter the pain. The abdominal aorta could be palpated easily. It appeared to be wider than normal especially at the level of the umbilicus. There was also an enlargement which was taken to be the right iliac artery.

FIG. 7.—The circulation in the right leg and foot was adequate.

Autopsy.—The aorta had formed a new aneurysm above the point of ligature. This sacculation prolapsed over the area which had been tied off. The tapes had cut through the vessel wall and had apparently eroded through the new aneurysmal sac into the small intestine, just beyond the duodenojejunal junction. The jejunum at this point had become attached to the aneurysmal sac. There had been massive hemorrhage into the small intestine. The old aneurysm was filled with a blood clot. There was thrombosis with organization in the left common iliac artery and in the old abdominal aortic aneurysm, the left external iliac artery and the left proximal hypogastric artery. The circulation to the stump had been carried by collateral enlargements of the lumbar and sacrospinal arteries and the distal left hypogastric artery. The right iliac arteries were intact and functioning well.

In addition, there was generalized and coronary arteriosclerosis. There were focal myocardial scars with cardiac hypertrophy and dilatation. Chronic pancreatitis and

chronic cholecystitis with cholelithiasis and mild cholangitis were present also. The kidneys had arteriosclerotic scars. There was an old focal encephalomalacia (Figs. 3 and 4).

This patient presented a problem for diagnosis on entry. His severe pain and anuria pointed to a urologic cause but examination did not seem to bear it out. Aneurysms of the abdominal aorta frequently have been regarded as urologic diseases, such as renal lithiasis, renal tumor, perirenal hemorrhage from trauma, hydronephrosis, or perinephric abscess. Uhle has presented an interesting review of this subject. It was possible to feel a mass in the left lower abdomen but pulsation in it was not elicited in the first days. There

FIG. 8.—The collateral circulation across the lumbar and sacral spinal arteries and through the distal hypogastric and its branches is beautifully demonstrated.

were no differences in the femoral pulsations noted. A retroperitoneal tumor was strongly considered. The roentgenogram showed some calcifications which were thought to be in the aorta or illiac vessels and suggested aneurysm to some members of the Department of Radiology. It was only after a progression of the symptoms and signs had led to an exploration that the diagnosis was definitely established.

The problem of controlling aneurysms of the abdominal aorta has not been solved as yet. When the aneurysm has had a traumatic etiology and the patient has youth and elastic vessels, operations such as those performed by Bigger have a happy outcome. But in individuals with aneurysms on an arteriosclerotic basis, with other calcified areas in the vessels, the chances for cure are much less favorable. Bigger emphasizes that aortic occlusion is unjustifiable in patients with extensive diffuse calcifications of the aorta.

Elkin claims that an efficient collateral circulation rarely exists about an aortic aneurysm. Consequently, occlusion should be partial only and not complete. Otherwise gangrene of the extremities will result. In case the collateral circulation has developed well, recurrence of the aneurysm in a short time is likely. Ligature of the aorta above the region of celiac or renal vessels cannot be tolerated as it jeopardizes the vitality of the intestines and kidneys. Ligation in continuity of a large artery will almost invariably be followed by gradual cutting through of the ligatures and either hemorrhage or reestablishment of the channel. If the proper time could be estimated, excision of the involved artery would seem to be a logical step unless an endo-aneurysmorrhaphy can be done.

It would seem that the method for occluding a large artery would be a relatively simple problem. But such is not the case. It has occupied the best thoughts of some of our most distinguished surgeons and still has not been completely solved. Pearse has reviewed the methods used and claims that all methods which depend upon external compression are dangerous. He has produced occlusion in animals by a spring screwed into the lumen of the aorta. This caused a gradual occlusion by thrombosis. It has not been found possible to easily insert such a spring in a deep wound. Extravascular occlusion by a sheet of cellophane has been successful in some animals but has been associated with rupture of the aorta in others. This method has promise and should be investigated more completely. At the time of Pearse's report the result of using a cellophane sheet was considered unpredictable. It has been successfully used since in the human by Harrison and Chandy to occlude a subclavian aneurysm. The method of occlusion of the aorta by rubber bands as advocated by Owings might also be considered. It has required multiple applications in order to get a successful outcome. This would be against its use for human cases.

Of all the external ligature material used to the present cotton tape seemed to be the most applicable. In Matas' patient a brilliant result was reported. The cotton tape was incorporated into the wall of the vessel and did not cut through. In four of the seven successful ligations it was the material used. And yet in our case, used according to the published methods, it has followed the rule and cut through the vessel walls producing a fatal outcome.

Our patient was of interest in the postoperative period because of the ensuing gangrene requiring amputation of the left thigh. The phantom limb from which he suffered was of extraordinary interest. Contrary to our usual experience we were able to define a trigger zone in his case. And by a simple redivision of the nerve trunks of the sciatic and lesser sciatic he was to a large extent relieved of his symptoms.

The postmortem studies of the collateral circulation were carried out by injection of a radiopaque liquid latex mass. This enabled us to obtain radiographs of the circulation and to dissect the vessels out intact afterwards. It was made possible through researches on this phase by Tobin. He showed a beautiful demonstration of the important collateral anastomoses through

the two lower lumbar and iliolumbar arteries to the left internal iliac (hypogastric) artery. This latter artery had slightly enlarged channels but its branches were normal in their distribution. The middle sacral and left lateral sacral arteries were filled with injection material and were easily dissected out. The left common iliac and the left external iliac arteries were completely occluded. No evidence of a previous rupture of the left common iliac artery could be demonstrated. The openings of the lumbar arteries into the aneurysmal sac were patent. All the arteries of the right pelvis and right extremity were filled with the latex material and appeared to be normal (Figs. 5, 6, 7, and 8).

Rupture of abdominal aortic aneurysm into the gastro-intestinal tract is a rare complication according to Smith. He found that there were, at most, probably less than 25 cases reported in addition to his own observation. In the present instance, the etiologic factor was that of a foreign body which would place this case in a different category.

We were much indebted to Dr. Stearns Bullen, Dr. Richard Jaenike and Dr. W. W. Scott, of the clinical branches; and to Dr. Charles Tobin of the Anatomical Department for his assistance in the vascular studies and dissections.

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THE TECHNICAL SIMPLICITY OF THE MATAS ENDO-ANEURYSMORRHAPHY*

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In Its Simplicity Lies Its Virtue

IN 1903, Dr. Rudolph Matas[1] gave to the surgical world an operative procedure designed for the surgical cure of aneurysm of the major peripheral arteries. Since his first reports on cases of arterial aneurysms cured by this method (aneurysmorrhaphy), there have been yearly reports on the successful cure of arterial aneurysms by the Matas method from surgeons throughout the world.

Although the simplicity of the technic of endo-aneurysmorrhaphy has been repeatedly discussed both by Doctor Matas,[1–4] and the author,[5–10] through the medium of journals and surgical meetings, it is unfortunate that its simplicity and applicability has not been thoroughly appreciated. A majority of surgeons think of this operation as being of a most formidable nature and resort to either the Japanese or English method of ligation and complete excision of the sac, or to a remote surgical procedure used by John Hunter in 1785.

The lack of familiarity with the over-all technical procedures of endo-aneurysmorrhaphy and its application, which not only involves the operative but the preoperative preparation and postoperative care as well, is due to two factors. First, the fear of uncontrollable hemorrhage during the operative procedure; and second, the inexperience of the surgeon in vascular surgery. The fear of uncontrollable hemorrhage during operations upon the heart and major arteries is dependent upon the dramatic descriptions of operations upon the heart and great vessels. It is common for the authors to dramatize the hissing noise and foaming blood in stab wounds of the heart, and the spurting stream of "whistling and gurgling" blood escaping from a major artery or aneurysm during operative interference. Then again, it has been almost a law that one, and only one, surgeon who has had his baptism in the blood of vascular surgery is allowed the honor and privilege to operate upon all the cases of injury or disease of the vascular tree. This unfortunate state of affairs has resulted in the training of very few vascular surgeons. It is the duty of all vascular surgeons to thoroughly train their residents in vascular surgery to the degree where they should handle all of the emergency injuries to the heart and major arteries. I have knowledge of some cases of stab wounds of the heart dying while waiting for the heart surgeon (the only heart surgeon!) to arrive and dramatically suture the bleeding heart. If, in the

* Read before the Southern Surgical Association, December 7–9, 1943, New Orleans, La.

past, we had taught the residents all the fundamentals, both physiologic and pathologic, pertaining to surgery of the vascular tree, we would have sufficient vascular surgeons who are now so badly needed at the battle fronts. I believe that many limbs could be saved today on the battle fronts if the surgeons who are doing the surgery had been thoroughly indoctrinated in all phases of vascular surgery while serving their residencies or apprenticeship. Therefore, the author requests that a course in vascular surgery be part of a surgical resident's training. This training should be conducted upon the experimental animal—fresh and preserved cadavers—and clinical application under a trained vascular surgeon, not one who limits himself to vascular surgery, but a general surgeon who has specialized in this most interesting and neglected field.

The operation of endo-aneurysmorrhaphy not only entails a thorough knowledge of the technic, but a familiarity with the pathologic physiology of aneurysms, both local and systemic, the absolute control of all bleeding during the operation, collateral circulation, methods of testing the collaterals and procedures used to develop a negligible and sluggish collateral circulation, and the preoperative and postoperative treatment.[1–10]

Doctor Matas divided his endo-aneurysmorrhaphy into the types: (a) Restorative; (b) reconstructive; and (c) obliterative.[2] The restorative is only applicable to the true saccular type of aneurysm. This type of aneurysm is produced mainly by trauma—stab wounds and gunshot wounds in which there has been only a small opening made in the artery—or in cases where there has been a minimum loss of the arterial wall. This type of aneurysm represents the minority group. This operation is one in which the lumen of the artery can be restored because there has been no loss of any part of the arterial wall. The restorative is not applicable to any other type or form of arterial aneurysm. Grave mistakes are made when adopting this procedure for aneurysms not of the pure saccular type.

The reconstructive operation was designed for the fusiform type of aneurysm. The technic consisted of forming a new segment of artery out of the walls of the aneurysmal sac. It was observed that in the majority of cases the newly formed arterial segment thrombosed very rapidly postoperatively. Therefore, this procedure was discarded in favor of the obliterative technic, which is recommended for all forms of arterial aneurysms except the pure saccular type. The obliterative endo-aneurysmorrhaphy consists of opening the aneurysmal sac with intrasaccular closure of all arterial openings and complete obliteration of the sac. Therefore, the obliterative type of endo-aneurysmorrhaphy is the procedure of choice in all arterial aneurysms both traumatic and organic of the peripheral arteries, with exceptions as noted above.

There is a group of arterial aneurysms in which the methods described above are not applicable. They are as follows: Common carotid group; subclavian group; aorta group; iliac group; aneurysms of the innominate artery, splenic artery, renal artery, common femoral artery, gluteal artery;

and the intracranial group. The procedure of choice in the above group is that of Anel, *i.e.*, proximal polar ligation of the main artery entering into the formation of the aneurysm. In the low carotid, subclavian and innominate groups, the author recommends the transpleural, transthoracic approach of Touroff.[13] The author[10] has used this method in one case with gratifying results.

In the above descriptions of the various technical procedures there is a single factor common to all, that is, the complete obstruction of the main peripheral artery at the site of the aneurysm. The one exception is the restorative technic used for the pure saccular type. However, the true saccular form is in the minority. That sudden occlusion of a main peripheral artery may result in grave and uncontrollable consequences should be common knowledge to all surgeons who undertake the cure of arterial aneurysms. Therefore, the surgical treatment of an arterial aneurysm should never be undertaken until the surgeon has unequivocally satisfied himself that there is present an adequate collateral circulation to maintain a normal metabolism to the tissues distal to the point of obstruction of the main peripheral artery.[8]

To determine the presence of an adequate or inadequate collateral circulation, there is only one test that will give accurate information regarding the presence or absence of collateral blood flow. This test was devised by Matas,[3–7] and is the only one that will obliterate the main artery proximal to aneurysm and concomitantly not interfere with the collateral circulation. This test is most simple in its application. It is performed as follows: A Martin rubber bandage is applied from the toes or fingers to a point just above the aneurysm. The Matas compressor[3] is now applied to the main artery above the rubber bandage. The compressor is screwed down until the main artery is obliterated. The Martin rubber bandage is now removed. One must be absolutely certain that the compressor does not slip. The foot or hand immediately after the removal of the rubber bandage has a cadaveric color. The blanched toes or fingers are now carefully watched for the return of a pink color. The time interval between the removal of the rubber bandage and the return of color determines the presence of an adequate or inadequate collateral circulation. If the time interval is over three minutes, the collateral circulation is inadequate.

If by the above test it is shown that the collateral circulation is deficient, then operation should be deferred until an adequate one is developed.

The development of an efficient collateral circulation can be obtained by two methods: The Matas,[3] or mechanical; and that of the author,[5–10] the physiological. The former is by the daily application of the Matas compressor which obliterates the main artery proximal to the aneurysm. This shunts the blood through the collateral vessels. The length of time the compressor is applied increases daily. The latter method (author's) is accomplished by blocking out the sympathetic ganglionated chain which supplies the vascular tree of an extremity with 1 per cent novocain or by surgical removal of the ganglionated sympathetic chain. This physiologic

method is simple in its application and results in a very rapid development of the collateral circulation. Since 1934, the author[5] has used this method extensively and its efficacy has been repeatedly demonstrated by others.[11–12] When it has been unequivocally demonstrated that an adequate collateral circulation exists, the patient is ready for operation. The next most important step is the absolute control of the blood flow through the field of operation. Unless the operation can be performed in an avascular field, the Matas endo-aneurysmorrhaphy should never be undertaken. If the blood supply both proximal and distal to an arterial aneurysm cannot be absolutely controlled, the aneurysmal sac cannot be opened. This is an inviolable rule of surgery of arterial aneurysms. Any surgeon who breaks this law will be immediately confronted with a most difficult situation and one that not infrequently ends in a catastrophe. There are two methods by which the operative field can be made avascular. The first, and most secure, is obtained by the application of a Martin bandage from the toes or fingers (only applicable to aneurysms of the extremities at least six to eight inches from the trunk) for a distance of eight to ten inches above the aneurysm. At this level a tourniquet is applied tightly around the limb. The rubber bandage is then removed leaving the operative field uncovered and completely avascular. The second method, not as secure as the first, is obtained by carefully dissecting out the arteries entering into the formation of the aneurysm, both proximal and distal to the aneurysmal, and applying temporary ligature (rubber catheters or rubber bands) to each vessel. In the majority of instances this is a very simple procedure because there is only the proximal and distal arteries to control. However, if the aneurysm is situated near the bifurcation of an artery, there may be more than the proximal and distal arteries to control.

When the above requirements are fulfilled, the patient is ready to be operated upon by the method of Matas, using either the restorative or the obliterative technic of endo-aneurysmorrhaphy. The type of anesthesia used can be either spinal or inhalation. The former is preferable for the lower extremities, whereas, the latter is superior for the upper extremities. The technical procedures of the operation (endo-aneurysmorrhaphy) are as follows:

After the anesthetic agent has produced its required effect, a Martin rubber bandage is applied to the leg or arm, beginning at the toes or fingers and extending the bandage above the aneurysm. A tourniquet is now applied above the bandage and the bandage is removed. The field of operation is thoroughly prepared and draped with sterile linen. An incision of sufficient length to give good exposure is now made over the aneurysm. The skin edges are protected with sterile towels and held in place by suitable towel clips. By sharp and gentle dissection the aneurysmal sac is now exposed. It is not necessary to completely expose the lateral walls of the sac. Being assured of perfect hemostasis, the sac can now be carefully opened. (It is important to examine carefully the relationship of the artery to the proximal and distal pole of the aneurysm before the sac is opened, because the sac

sometimes rotates on its polar axis and the proximal artery will run along the posterior wall of the sac. This artery may be injured when the sac is opened or the opening of the proximal artery into the aneurysmal sac will be difficult to locate.) The sac is now gently cleaned of its contents which consists of laminated clot and fluid blood. A diligent search is now made for the openings of the artery or arteries into the lumen of the sac. When they are identified a catheter, which has been previously anointed with sterile liquid petrolatum, is introduced into the openings. This is to determine their patency and to see if they connect with an artery. The catheter is discarded and the openings are closed with interrupted sutures of silk or cotton. The needle should take good firm bites through the lips of the openings. When all openings are closed the tourniquet is loosened to test the efficiency of the closure of the openings. This step is absolutely essential before the sac is obliterated, because if the openings are not thoroughly closed, a hematoma will form or another aneurysm may develop. If there is no bleeding from the sutured openings (occasionally a few extra sutures may be necessary to control oozing from the sutured openings), the aneurysmal sac is obliterated by layers of interrupted sutures of silk or cotton. These sutures should bring the walls of the sac together. The superficial incision is now closed and a pressure dressing is applied to the wound.

By using the above technic there has been no disturbance or interference with the vessels belonging to the collateral circulation. The vessels that are in juxtaposition to the sac of the aneurysm are likewise not disturbed—a most important and frequently overlooked detail when other surgical procedures are used.

The extremity is enclosed in two or three layers of cotton batting. Heat should not be applied to the extremity as it has been shown that heat increases the demands for greater vascular supply and if not forthcoming, there results varying degrees of anoxia which may bring about tissue destruction and necrosis. If the collateral circulation is adequate and is maintained, there should be no fear of ischemic necrosis of the extremity.[7] If ischemic necrosis does occur, it is indicative of poor preparation of the patient for the operation.

The patient is kept in bed for 10–15 days postoperatively, then allowed up, and gradually returns to his or her former activities.

CONCLUSIONS

(1) Endo-aneurysmorrhaphy is the surgical procedure of choice in treating aneurysms of the peripheral arteries.

(2) There are only two types of endo-aneurysmorrhaphy that are applicable to aneurysms of the peripheral arteries: The restorative (saccular) type; and the obliterative (fusiform) type.

(3) An adequate and sustained collateral circulation must be present and satisfactorily demonstrated before the operation is undertaken.

(4) The collateral circulation can be developed by two methods; Me-

chanical (Matas method); and physiologic (blocking out the sympathetics, the author's method).

(5) Absolute hemostasis must be obtained and continued until the intrasaccular toilet is completed.

(6) This operation is contraindicated in all arterial aneurysms in which the blood supply cannot be unequivocally controlled during the entire operation.

(7) It is the simplest of all surgical procedures used for the cure of arterial aneurysms.

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DISCUSSION.—DR. HERMAN E. PEARSE, Rochester, N. Y.: I have had very little clinical experience in aneurysms but thought it might be of interest to review briefly the matter of experimentally occluding the larger arteries. The three different methods of attack in occlusion of the arteries are by external compression; by efforts to produce scar in the wall; and by internal occlusion.

External compression may be done by the Halsted band, the Matas band, the Neff clamp, or by various types of ligatures of silk, catgut tapes, or fascia. Doctor Halsted said that he could not accurately judge the tension necessary in the use of ligatures or tapes. I was sorry Doctor Matas did not have the opportunity of telling more about his band, for it is very useful. There are two other methods not illustrated, one, that of Doctor Matas, where he did plication of the aortic wall; another the elastic ligature of Owings. Doctor Mont Reid is responsible for the statement that external pressure on the aorta will invariably result in atrophy and death of the vessel wall.

The second method is by fibrosing the wall with diathermy, by injection of sclerosing solutions into the wall or by cellophane. Three years ago I reported experiments on the fibroplasia caused by wrapping cellophane about the aorta of dogs. In this I mentioned that in two of the 24 experiments there was no reaction. Subsequently, in another

series, a large number showed no response, the cellophane appearing inert in the tissues while in other animals an intense fibrosis occurred which shut off the vessel. Thus far, this discrepancy has not been explained but it is suspected that there is some substance in the cellophane, the presence or absence of which determines the amount of fibroplasia produced.

The third principle is by internal thrombosis. Doctor Mont Reid was the first to use this by means of placing a fascial plug with a tube in its center into the artery. Many years ago, while working in the Hunterian Laboratory, in Baltimore, I reviewed this subject, and came to the conclusion that internal occlusion offered the best means of obstructing the aorta. I attempted to insert metal tubes into it so as to cause gradual thrombosis, but found this difficult technically. So a tube was made of a coiled spring that could be screwed into the vessel through a puncture wound. This was successful, but has its disadvantages, for I find that one gets out of practice and hemorrhage occurs, so I do not advocate it as a final procedure. However, I do believe it demonstrates a principle which, if followed with better technical methods, may accomplish the result. I have returned to the opinion held 17 years ago that the aorta must be shut off from within.

MAJOR DAVID T. MONAHAN, Bridgeport, Conn.: This is a brief report of a case of aneurysm of the terminal aorta treated by proximal ligation in stages, after the method described by Owings, in 1941.

Case Report.—The patient was a Negro, age 49, who complained of abdominal pain and weight loss of 20 pounds. He had been under treatment for syphilis; the Wassermann test was positive. Physical examination disclosed a fixed, pulsating tumor just to the right of the umbilicus. He was operated upon in three stages, three weeks apart. At the first operation, March 20, 1942, the aneurysm was found to extend from about 1.5 inches below the renal arteries to the bifurcation of the aorta. An area about the size of a dime on the anterior surface was paper-thin. A rubber band, three-fourths of an inch wide, was placed proximal to the aneurysm and sutured in the shape of a funnel, so that about 75 per cent of the circulation into the aneurysm was interrupted. There were no circulatory symptoms after this procedure and the patient was out of bed in two weeks.

At the second operation a rubber band, one-half inch wide, was placed just below the site of the previous band, proximal to the aneurysm, and the circulation was shut off entirely. Postoperatively, his right leg was progressively cooler from the knee to the toes and there were fibrillary twitchings in the calf muscles of this leg, although he could move both legs without difficulty. These symptoms cleared up in a week except for some coldness in the right foot. He was allowed to go home for a week between the second and third operations.

At the third stage it was found that there was no circulation through the aneurysm. Two heavy silk ties were placed below the rubber band; both common iliacs were divided and ligated and the inferior mesenteric was ligated. Postoperatively, the patient again had coldness in the right leg and fibrillary twitchings for a week. He had considerable pain in his foot, which persisted.

He was discharged from the hospital in two weeks, was up and around and had no difficulty walking. The aneurysm could be felt only as a shrunken, firm mass. He complained of pain in his right foot persistently, and the advisability of paravertebral block was being considered when he had a massive hemorrhage into the gastro-intestinal tract, and died August 15, five months after the first operation.

Autopsy disclosed that a perforation had occurred into the third portion of the duodenum at a point just proximal to the proximal rubber band. Section through this area showed the aneurysm to be filled with old organized clot and at the area of ligation there was a band of tissue of cartilaginous consistency, without any vestige of a lumen. The silk sutures were found imbedded in the lower end of this band. The rubber was not apparent grossly, and was presumably fused with the band of scar tissue, although the pathologist was unable to identify it. To the right of the aorta just proximal to the constriction was a new aneurysm filled with clot which communicated with the perforation.

The heart and kidneys were normal. Section of the aorta showed mild syphilitic aortitis.

DR. JOSEPH E. J. KING, New York City: I feel that we all regret very much that Doctor Matas did not have more time, and I hope he will have more to say in closing his discussion.

I shall limit my remarks to occlusion of the internal carotid artery in nine cases of intracranial arteriovenous fistula and two cases of intracranial aneurysm. Not having had access to Doctor Matas' band, and knowing of several cases in which death had occurred almost suddenly following ligation of the common carotid artery, I have employed the following method: To take the place of the Matas' band I have used ordinary skin clips, two being used and placed close together so that the artery would not so likely be cut through. The small sharp tips are broken off the clips, and then filed down. The clips are bent in the form of *omega*. A piece of fine plain catgut is tied in one of the eyelets of the skin clips so that in case quick removal is necessary it can readily be done. If there is no need for removing the clips, the catgut dissolves and the clips remain in place.

We have placed the two skin clips bent into the form of *omega*, around the internal carotid artery just beyond the bifurcation of the common carotid, and closed the skin clips down on the vessel until only the faintest bruit could be heard with a stethoscope placed directly over the eyeball. The intention is to shut off about three-quarters or four-fifths of the lumen, but not completely to occlude the lumen.

In the first two cases we reopened the incision in order that we might completely occlude the artery by further compression of the clips, but this was found to be unnecessary. In one instance the clips had completely and slowly divided the vessel, and both ends of the vessel were found to be firmly thrombosed. In the second case the vessel was almost cut through by the clips so that when the clips were more firmly and completely compressed, they cut through the remaining portion of the vessel without bleeding, *i.e.*, both ends had become firmly thrombosed. Therefore, in subsequent cases it has not been found necessary to completely close the clips at the second stage, and it is believed that complete thrombosis takes place slowly over a period of two or three weeks. At any rate, application of the clips in this manner succeeded in obliteration of the bruit and recession of the eyeball. Should it be necessary, the external carotid artery can be ligated and divided at the same time.

There were no deaths. One patient, a woman, had weakness in the opposite side for six or seven hours, but with elevation of the foot of the bed, no further weakness developed, and the weakness in the opposite side cleared up.

In the majority of cases the clips have remained in position, but in two or three cases they worked out to the surface and were readily removed with a thumb forceps. No damage has occurred to the vagus nerve or the jugular vein.

DR. JOSEPH A. DANNA, New Orleans, La.: I am very glad of the opportunity to lend my support to Doctor Gage's argument that the Matas operation for aneurysm is simple. Most articles written on this subject do not give the impression that it is a simple operation. Even what has been told you this afternoon I am sure does not leave that impression with you as clearly as I would like.

I have two criticisms to make of Colonel Gage's presentation so far as showing how simple it is. In the first place he made the incision two or three times as long as necessary. In the second place it is not necessary to obliterate the sac. There are some instances in which you cannot obliterate the sac. I have only done so in the first two or three cases in which I tried to do a restorative operation. The operation is very simple. You make an incision over the most superficial part of the sac. You can make it shorter than the length of the sac but never longer. Be sure you do not cut some important structures as you go down to the sac. After that you cannot injure anything. If you open the sac wide and cannot find any arterial opening you will probably readily find the openings by everting the edges of the incision and looking for them in the superficial wall of the aneurysm near the incision line. Once you have the sac open and the openings located it is very simple to put in a few sutures, as stated.

DR. THOMAS B. AYCOCK, Baltimore, Md.: I realize that Doctor Gage was speaking of aneurysms of the periphery rather than those which occur in the chest. Recently, however, I had one of the innominate artery, and, more recently, one of the subclavian just distal to the innominate. I notice these have not been referred to and will relate briefly my experience. The first, I was not able to isolate. In retrospect, I wonder if the approach I used is not out of date. Both were approached anteriorly through the superior mediastinum. I have ligated the subclavian in its second part following accidental

injury to it by the posterior extrapleural approach, namely, by resecting the third, second and first ribs on the right, and I now believe it a better route to reach either the innominate or subclavian than the sternoclavicular disarticulation, which method I employed. I would like to hear what Doctors Matas and Gage have to say in this particular, if they have had any experience with aneurysms of this type.

Dr. Alfred Blalock, Baltimore, Md. (closing): I am sure you will all agree that it is a real privilege to hear Doctor Matas describe a patient operated upon by him 55 years ago. The fundamental principles regarding the treatment of aneurysm enunciated by Doctor Matas, in 1888, still holds.

I am sorry there was not time for Dr. Duane Carr, of Memphis, to describe a patient with an aneurysm of the thoracic aorta who was operated upon by Dr. John Alexander, of the University of Michigan, several months ago. The operation consisted of ligation of the thoracic aorta and the removal of the aneurysm. The fact that the patient withstood the complete occlusion of the thoracic aorta suggests that there might have been a coarctation of the aorta associated with the aneurysm. It would seem that there must have been some added incentive for the formation of collateral pathways, for it is well known that patients with thoracic aneurysms will not usually withstand ligation of the thoracic aorta. Doctor Carr informs me that this patient has a rather marked hypertension at the present time, and it might be that a procedure such as Doctor Park and I described today (or modification of it, such as Doctor Matas mentioned) should be considered.

The work of Dr. Arthur Blakemore, of the Presbyterian Hospital, New York City, constitutes, in my opinion, one of the greatest advances that has been made in the treatment of aneurysms. This work has not received the attention which it merits. By the use of the wiring technic which he has described, one is able to cause an obliteration of aneurysmal sacs which cannot be attacked by other methods. It is my opinion that this procedure should be more generally adopted in the treatment of aneurysms of the thoracic and the abdominal aorta.

Dr. John J. Morton, Rochester, N. Y. (closing): I have nothing to add, and I would welcome it if Doctor Matas would give us a little more information in his closing remarks. I came here to learn.

Dr. Rudolph Matas, New Orleans, La. (closing): How to relieve the grave effects of the congenital stricture or stenotic coarctation of the aorta caused by developmental defect, by creating a new blood channel which will bridge over the obstruction and restore the continuity and volume of the aortic stream, is one of the new, but still distant, problems of intrathoracic surgery which Doctors Blalock and Park have ingeniously, and with great perseverance and skill, attempted to solve. It is a fitting example of the type of operations which would have been dismissed as purely chimerical, or outside the domain of practical or rational surgery, 25 years ago, but have now become tangible as experimental and clinical realities.

In the present experimental inquiry, the authors, particularly Doctor Blalock,* have brought to its performance the benefit of a previous experience in aortic surgery by his studies on the "Effects of Complete Occlusion of the Thoracic Aorta, *etc.*" In the present investigations the authors have had to deal with two problems as these apply to the dog: (1) The creation of a stricture, coarctation or total occlusion of the thoracic aorta in imitation of the congenital strictures in man; (2) the creation of a blood channel, or arterial duct, to connect the proximal with the distal side of the obstruction, thereby restoring the blood stream to the distal side. In the dog, this means two problems to be dealt with; in man, only one, since the stricture or obstruction already exists and the only problem is how to restore the continuity of the aortic stream.

Experimentation and clinical experience with various methods of ligation since the days of Astley Cooper, who first (1817) ligated the abdominal aorta in man, have shown that the *sudden total* occlusion of the aorta in normal individuals is fatal, and that the period of survival is generally directly proportional to the proximity of the ligature to the heart. It is only in individuals who have suffered from a long-standing obstruction

* Jour. Thoracic Surg., 2, No. 1, 69–75, October, 1932.

in the aortic stream (*i.e.*, aneurysm) that a total occlusion may be followed by survival, solely because the collateral circulation has had a chance to develop.

In the same way it is now well established that the gradual occlusion in several stages of the aorta throughout its tract from the subclavian to the bifurcation, is compatible with survival (Halsted, Matas, C. Allen, Reid, Pearce, Owings, *et al.*). Even the total, but gradual, obliteration of the great trunks that spring from the aortic arch (innominate, carotids and subclavian) is compatible with life, if time is given for the development of the collateral circulation by way of the intercostals.

In their experiments, the authors have aimed at an immediate and complete occlusion of the aorta at the so-called isthmus of the arch, immediately outside the subclavian, which is the usual seat of the congenital occlusion in the so-called "adult" type (Bonnet) in postnatal life; *i.e.*, a few millimeters from the level of the obliterated ductus arteriosus.

I need not rehearse the steps of the operation which Doctor Blalock has so well described and illustrated. It is well to note, however, that our authors tried in their preliminary experiments to obtain the occlusion of the aorta by Owing's method of gradual obliteration in three stages, in which the artery was compressed progressively with a rubber band shaped like an hour-glass.

In 1940, when, through the courtesy of Doctor Owings, I first described his procedure at the meeting of the American Surgical Association,* I believed that the use of a rubber band shaped like an hour-glass was a valuable suggestion, as the elastic rubber would yield to the impact of the aortic stream as this entered the hour-glass in a conical shape, thereby diminishing the tendency to atrophy of the aortic wall at the proximal (cardiac) margin of the band.

The admirable results obtained by Doctor Owings in his experimental occlusions of the thoracic aorta fully confirmed the theoretic advantages of the method, and my own belief that the major difficulties in the control of the aorta had been overcome. But the unexpected outcome of the remarkable operation for an ilio-aortic aneurysm, which Major Monahan has just read, and of which I had been previously informed through his courtesy, made me realize that what seemed to be a simple and safe procedure had also its pitfalls and fallacies when performed in pathologic human subjects. For, in the operation reported by Major Monahan (the occlusion of the aorta for an aneurysm of the common iliac, at the bifurcation), the application of the Owings' band to the aorta in three sittings had accomplished its purpose in controlling and suppressing the aneurysm without apparent complications. The patient, apparently well, had returned to his regular mode of life when suddenly he died as the result of a profuse and uncontrollable gastro-intestinal hemorrhage. At the autopsy, it was found that death had been caused by the rupture of a small aneurysm which had developed in the aorta immediately above the edge of the rubber band on the proximal side of the band. The sac had contracted adhesions with the duodenum into which it ruptured with fatal results. The aorta had been thinned and stretched into an aneurysmal sac by the pounding of the aortic current against the upper edge of the rubber band. Thus it was, that what seemed to be a most perfect and brilliant success proved, in the end, to be an unpredictable and disastrous failure. The experience of Doctors Blalock and Park with a procedure very similar to that of Doctor Owings was complicated by hemorrhages at the seat of the constriction more often when the constriction was increased at the second stage of the procedure. This led the authors to adopt the original procedure which Doctor Blalock has just described.

The vital step in this operation is the successful transplantation of the subclavian artery to the distal segment of the divided aorta. Failure to keep this channel in functional activity means the prompt death of the animal. Thrombotic occlusion is also fatal, as five cases seemingly ended in this way.

As the anastomosis between the proximal aorta and the distal segment is effected by an end-to-side anastomosis of the subclavian to the aorta, the liability to mechanical occlusion by the kinking of the subclavian channel at the line of suture makes it more liable to occlusion than a straight end-to-end anastomosis. The vital importance of this anastomotic subclavian bridge is well demonstrated by the fact that the general arterial pressure which is uniformly sustained as long as the subclavian channel is functioning, becomes unbalanced when for any reason the subclavian channel is blocked.

* ANNALS OF SURGERY, 112, 909–922, November, 1940. *Ibid.*: 115, 596, 1942.

Then the blood pressure in the head and vascular region supplied by the branches of the aorta above the line of aortic section, begins to rise progressively while the blood pressure in the territories supplied by the aortic branches of the lower segment, begins to drop progressively and fatally. In this way, the experimental coarctation of the aorta reproduces the condition that prevails in the clinical cases of marked congenital aortic stenosis in which the hypertension in the vessels of the brain and upper extremities and the hypotension in the lower extremities and vascular areas below the aortic stenosis are distinctive characteristics.

That the incidence of these coarctations is far more frequent than is estimated in the general statistics of postmortem findings (about 1 in 2300, Fawcett, 1902; 1 in 1500, Bramwell and King, 1942) is the opinion of observers such as Liebman, who was especially interested in finding them. The more frequent clinical discovery of this anomaly is also in accord with our experience with other disorders which have become far more frequent since roentgenologic and other more accurate ways of clinical investigation have made them more apparent and recognizable (patent ductus arteriosus, bronchogenic carcinoma, aortic aneurysm, appendicitis *etc.*).

One of the interesting questions brought out by Blalock and Park is the fact that after the occlusion of the aorta by their operation, the general blood pressure gradually rises from the relatively low hypotension at or shortly after the operation to an increasing hypertension in the carotid and femoral tracts, which progressively follows in from four to ten months after the operation. The reasons for this progressive systemic rise in the blood pressure are not clear, despite the assumption that the general hypertension is a reactionary response to renal ischemia caused by the lowered vascular supply of the kidneys and other organs below the experimental coarctation, as suggested by Goldblatt's theory of hypertension following a partial occlusion of the renal arteries.

The fact that more than 23 per cent of the animals have survived this formidable, but very skilfully planned, operation is in itself a great compliment to the skill and perseverance of the operators. The complications from technical defects (thrombosis, hemorrhage) are presumably avoidable, but, thus far, the one complication that seems to be specifically peculiar to the total acute experimental occlusion of the aorta in dogs, is paralysis of the hind quarters, which occurs "in about 50 per cent of the animals, varying from a short transitory weakness of the legs to a more or less lasting spastic disability in a few." While this is related to an ischemic disturbance in the circulation of the lumbar cord and cauda equina, the mechanism by which this occurs still awaits a clear explanation.

In eight of the animals operated upon by the authors, the cause of death was not discovered, showing that death may occur after this operation in spite of apparent good technic and physiologic restoration of the aortic flow.

There can be no question that many minor aortic coarctations of large caliber exist without causing appreciable disturbance in the life of the individual. While it is universally recognized that the effect of a stricture of narrow caliber is, in general, to shorten life and is liable to cause sudden death from cerebral hemorrhage, dilatation of the heart, ruptured aneurysms of the ascending arch of the aorta, and other effects of the progressive hypertension in the vessels proximal to the obstruction, it is also true that life is compatible with even advanced old age, despite a practically complete occlusion of the aorta at the coarctation (92 years, Reynaud), the patient's life expectancy largely depending upon the development and efficiency of the compensatory circulation through the intercostal collaterals. In view of this compensatory circulation, the indications for such grave and adventurous operations as that suggested by the authors' experimental study would be very rare indeed, even granting the feasibility in the human subject.

GENERAL CONCLUSIONS

It would seem from the evidence gathered in this carefully and skilfully conducted experimental study:

1. That the operation devised by the authors for the relief of congenital coarctations, stenoses or atresic obstructions of the thoracic aorta is technically feasible, and has

proved successful in accomplishing its purpose in a fairly large proportion of the dogs in which the operation was performed, (10 of 43, or more than 23 per cent).

2. That the experimental study devoted to the practicability of producing an arterial blood conduit to bridge over a permanent obstacle in the aorta is a valuable contribution to the technic of experimental aortic surgery, and the study of the secondary hypertension following in the wake of this operation is another valuable contribution that has come out of this study.

3. Unfortunately, the operation, as applied successfully on dogs by the authors, is not applicable to human subjects, as admitted by the authors, because the anatomy of the subclavian in man does not permit of the transplantation of this artery from its extrathoracic cervical bed to the aorta without fatal damage to its important cervical collaterals, besides the permanent damage inflicted on the main arterial supply of the arm.

4. The use of the carotid as a substitute for the subclavian for the same purpose would involve serious difficulties in the transplantation of that artery from the neck to the thoracic aorta. In addition to the objections stated by the authors, there is the liability of cerebral complications which add to the gravity of the procedure and make its performance scarcely justifiable except as an adventure in desperate cases.

5. That in attempting to relieve the sufferers from the effects of a congenital coarctation of the thoracic aorta in which the essential or fundamental problem consists in restoring the continuity of the aortic stream, a much simpler and presumably less hazardous procedure would be to divide the aorta at the seat of stricture and connect the divided segment by a vitalium tube lined by an autogenous venous graft, in the manner suggested by Blakemore, Lord, and Stefko.*

6. While the experimental evidence thus far available is in favor of the practicability of veno-vitalium grafts, the further study of this procedure as permanent blood channels in this connection, is desirable in an effort to simplify the attainment of the, at present, very important but difficult objective, namely, the permanent repair and functional restoration of the larger damaged arteries.

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ANNOUNCEMENT

AN ARMY EVACUATION HOSPITAL at Charlottesville, Va., organized under University of Virginia sponsorship, has celebrated the completion of its first year of overseas service by presenting to the University of Virginia a set of surgical instruments which are exact reproductions of instruments used 2000 years ago and found in the ruins of ancient Pompeii and Herculaneum.

Three boxes containing the 57 historic reproductions have been received by President John Lloyd Newcomb and have been turned over to Dr. Harvey E. Jordan, Dean of Medicine. They will be placed on exhibition in the library of the medical school.

Original instruments from which the reproductions were made are in the national museum at Naples. The instruments were found in the ruins of the cities buried in the eruption of Mount Vesuvius in 79 A.D., and most of them were in use long before that date.

Cavalier Gulielmo Callo made each of the reproductions by hand from the metal originally used, usually bronze. They have been antiqued to look like the original by a secret process.

Lieutenant-Colonel Staige D. Blackford, member of the university faculty and chief of medical service of the Evacuation Hospital, has sent the gift with an explanation that it was made possible by contributions from doctors, nurses and administrative staff members.

President Newcomb has written in appreciation, assuring the donors that their gift will serve "to stimulate interest in the history of medicine on the part of future generations of University of Virginia medical students."

Ancient surgical instruments in the set of reproductions include a variety of forceps, speculum, curettes, probes, blood cups, hooks, scissors in several sizes, irrigating canula, spatulas, cauteries, needles, knives and bistouries, a tongue depressor, a tenaculum for molars, *etc.* There are two historic pictures in the collection.

The evacuation hospital was organized in the Winter of 1942. It served in North Africa.

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The publishers of the ANNALS OF SURGERY regret the late appearance of this issue. Shortages of manpower, the busy schedules of contributors and other factors beyond our control made it impossible to publish on the usual date.—The Editors.